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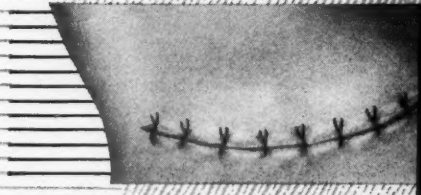

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THE
AMERICAN
SURGEON

Vol. 19, No. 6 June, 1953

NEUROGENIC TUMORS OF THE ANTERIOR AND MIDDLE MEDIASTINUM

A REPORT OF TWO CASES

COMMANDER L. E. TEBOW, MC, USN*
CAPTAIN R. B. BROWN, MC, USN*

Bethesda, Md.

The characteristic location of mediastinal neurogenic tumors is deep in the posterior compartment. Rarely are these neoplasms found in an anterior or middle position. In 1944, according to Kent, et al.,[15] only 5 of the 91 mediastinal neurogenic tumors reported to that date were located anteriorly. Of these, 1 reported by Cabot[7] must be excluded. Neither in the original article by Cabot nor in the review is there conclusive evidence that the tumor is of neurogenic origin. Since this excellent review by Kent and associates there have been an additional 126 mediastinal neurogenic tumors reported.[1, 3, 4, 5, 6, 8, 9, 10, 11, 12, 13, 14, 16, 17, 18, 19, 20, 21, 23] Only 5 of these were found in an anterior or middle position (table 1).

The following 2 cases are the subject of this report. One of these, a benign cystic neurilemmoma, was located in the middle mediastinum and, because of its location and gross appearance at operation, was erroneously diagnosed as a bronchogenic cyst. The second case is a malignant neurilemmoma (Schwannoma), a solid tumor, located in the anterior mediastinum.

Case 1—Mrs. E. R. N., aged 60, white woman, was admitted to the U. S. Naval Hospital, Bethesda, Maryland on March 21, 1952. She was received as a transfer patient from another institution where she had been studied because of complaints of soreness in her chest and severe postprandial pain located at the level of the xyphoid process. The latter pain radiated into her back and up into her neck. Except for a partial thyroidectomy in 1946 and a mild hypertension (160/110), the history and physical examination were essentially negative.

Laboratory studies, including non-protein nitrogen, blood cholesterol, serum

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protein, albumin-globulin ratio, phenolsulfonpthalein test, Kahn, complete blood count, urinalysis, and basal metabolic rate were within normal limits. Radioactive iodine (I^{131}) uptake study demonstrated a 24 hour concentration over the thyroid of 13 per cent and a 48 hour concentration of 10 per cent, with no uptake over the chest. Chest roentgenograms and fluoroscopy revealed a mass in the middle mediastinum. The mass was described as 6 to 8 cm. in diameter, nonpulsating, and located to the right of the heart. A barium swallow demonstrated displacement of the esophagus posterior and to the left. The roentgenologic diagnosis was pericardial or bronchogenic cyst (fig. 1).

Bronchoscopy revealed slight compression of the inferior portion of the two main bronchi just distal to the carina. Esophagoscopy showed nothing abnormal.

Clinical impression: Mediastinal tumor, probably a bronchogenic cyst.

On April 9, 1952 a right exploratory thoracotomy was done. Through a posterolateral incision the sixth rib was excised and the pleural cavity was opened. The

TABLE 1

Author	Date Reported	Position	Type*
1. Hosoi	1931	Ant. Sup.	Sarcomatous transformation of a known neurofibroma.
2. Cutler & Gross	1936	Ant. Sup.	Neurofibroma.
3. Kent, et al	1944	Ant. Inf.	Ganglioneurosarcoma.
4. Kent, et al	1944	Ant. Sup.	Neurogenic sarcoma (Reported by Ackerman[1] as neurilemmoma).
5. D'Abreu	1947	Ant.	Ganglioneurofibroma.
6. Mandeville	1947	Mid.	Neuroblastoma.
7. Ackerman & Taylor	1951	Ant.	Ancient neurilemmoma.
8. Sabiston & Scott	1952	Ant.	Neurogenic sarcoma.
9. Sabiston & Scott	1952	Ant. Inf.	Neuroblastoma.

* No attempt has been made to group these tumors according to one classification. It is believed that rather than cause further confusion in the pathologic nomenclature they should be listed as classified in the original article.

cyst was easily demonstrated. It was projecting from the right mediastinum with the junction of the azygos vein and the vena cava at its upper pole. The mediastinal pleura was incised over the tumor in a line anterior to the course of the vagus nerve. The cystic mass was easily shelled out of the mediastinum by blunt dissection. Anterior and lateral displacement of the right main stem bronchus and the posterior displacement of the esophagus by the tumor were demonstrable during its removal.

The lung was re-expanded, water seal drainage was established through a catheter placed in the eighth interspace at the posterior axillary line, and the wound was closed in layers.

The tumor excised was a cyst, well encapsulated, approximately 8 by 10 cm. in size, containing straw colored fluid. The wall measured about 3 mm. in thickness and was trabeculated, but there were no true loculations. Impression: bronchial cyst.

The postoperative course was uneventful and the thoracostomy tube was removed on the third postoperative day. The patient was symptom free and discharged to her home 10 days after surgery.

Pathology report. Gross examination: The specimen consists of a formalin fixed "bronchial cyst" measuring 6 cm. in diameter, the exterior surface of which is rough and friable. The inner surface is lined by a smooth glistening membrane. On one cut section the wall has a yellow-gray color and almost cheesy consistency.

Microscopic examination: Microscopic study shows a cellular tumor mass that has a broad acellular capsule. There are wavy bundles of fusiform cells which in some areas have a palisading arrangement around hyaline centers (Verocay bodies). The nuclei are vesicular and vary from round and plump to fusiform and

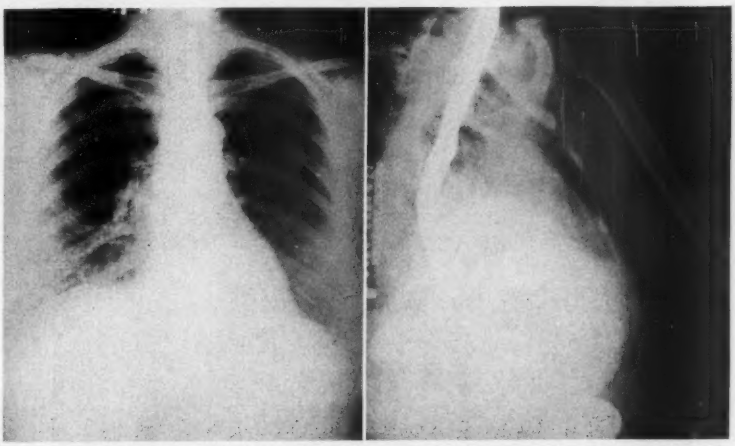

FIG. 1, Case 1. Postero-anterior and right anterior oblique roentgenograms showing the position of the tumor. The barium swallow shows the posterior displacement of the esophagus.

spindly. Gathered throughout there are clumps of foam cells. It is also noted that there are many large vascular spaces through the tumor that have, in many instances, thick hyalinized walls.

Diagnosis: neurilemmoma (fig. 2).

Case 2—Mr. A. J. G., aged 48, was admitted to the U. S. Naval Hospital, Bethesda, Maryland on Aug. 5, 1952 for study of an intrathoracic tumor demonstrated by chest roentgenograms incident to routine physical examination which was made one month before. He had no respiratory or other systemic symptoms, and stated that he felt perfectly well. The physical examination was essentially negative. The blood count, urine, Kahn, sedimentation rate, and albumin-globulin ratio were normal. Histoplasmin and coccidioidin skin tests were negative. The tuberculin test (P.P.D. intermediate) was positive. A chest roentgenogram demonstrated a well circumscribed lesion in the anterior mediastinum

extending to the right hilus (fig. 3). The electrocardiogram was normal. Bronchoscopy was negative, and cytological studies of the bronchial washings revealed no tumor cells.

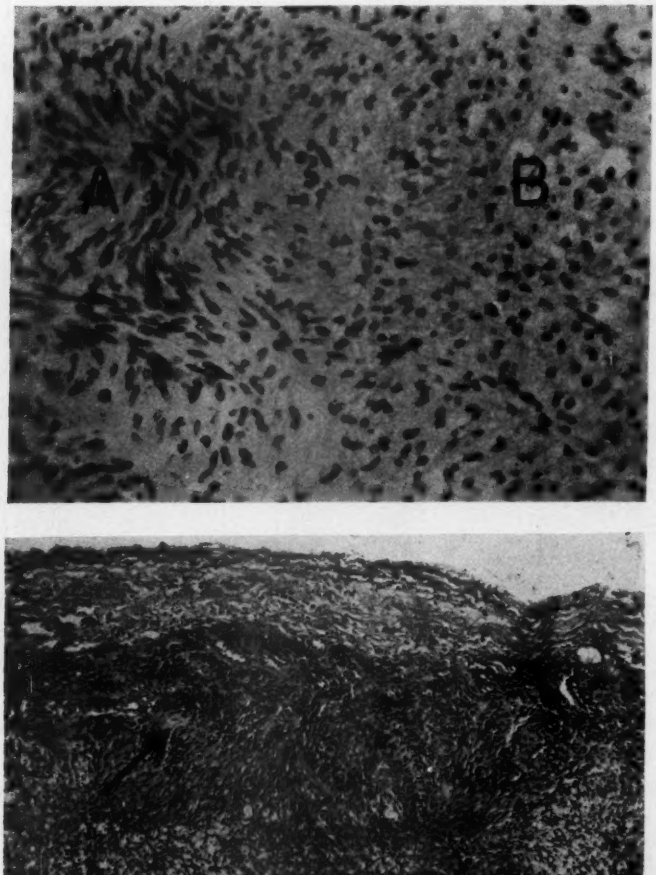

FIG. 2, Case 1. A. Section showing both Antoni "A" and "B" tissue. Typical of neurilemmoma. B. Section of tumor under lower power showing Verocay bodies.

Exploratory thoracotomy was done on Aug. 19, 1952. The right chest was entered through a posterolateral incision with resection of the sixth rib. There was an irregular tumor projecting from the anterior superior mediastinum,

measuring about 6 by 8 cm. The mass was lobulated, firm, well encapsulated, and with no gross evidence of invasion. The mediastinal pleura was incised over the tumor, and the well circumscribed tumor shelled out by blunt and sharp dissection. A vascular pedicle running cephalad from the mass was cut and ligated, and the tumor was removed. The lung was re-expanded and water seal drainage was established through a rubber catheter brought out through the tenth interspace in the midaxillary line. Penicillin and streptomycin were instilled into the chest cavity and the wound was closed in layers.

Operative and frozen section diagnoses were deferred.

The patient's recovery was uneventful. The thoracostomy tube was removed on the second postoperative day and the wound healed per primum. He was discharged to full duty on Oct. 15, 1952.

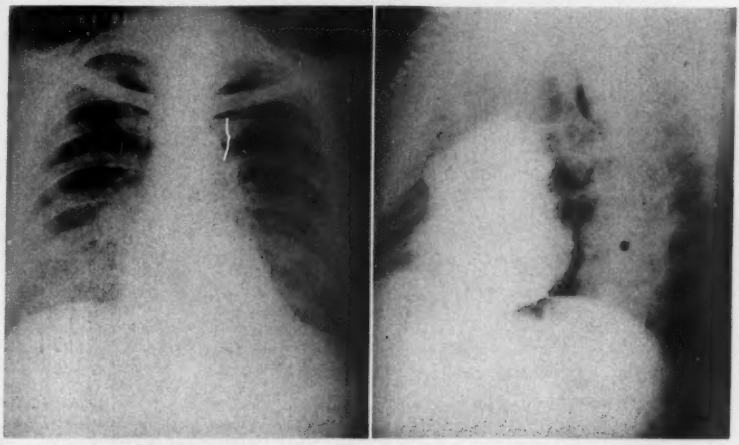

FIG. 3, Case 2. Postero-anterior and left anterior oblique roentgenograms showing the anterior position of the tumor.

Pathology report. Gross examination: The specimen consists of an irregular spherical mass measuring 9 cm. in diameter. The external surface presents a smooth membrane with attached globules of fat. Section reveals firm small lobules which in some areas contain yellowish and brown streaks. The tumor shows a marked tendency to break into small fragments.

Microscopic examination: Microscopic study shows a very cellular tumor that is separated into lobules by broad bands of hyalinized connective tissue. The structure of the tumor shows interlacing and parallel bundles of fusiform cells similar to those seen in neurilemmomas; however, the individual cells are pleomorphic and range from irregular plump cells to cells with long and spindly processes. Predominately the nuclei are vesicular with fine chromatic granules. There are also many degenerating cells with small pyknotic nuclei. The reticulum stain shows a sparse scattering of thick reticulum fibers, while Bielschowsky's stain shows an abundant number of fine fibrillary nerve processes. This picture is that of the tumor described by Stout as a malignant schwannoma.[24]

Diagnosis: Malignant schwannoma (fig. 4).

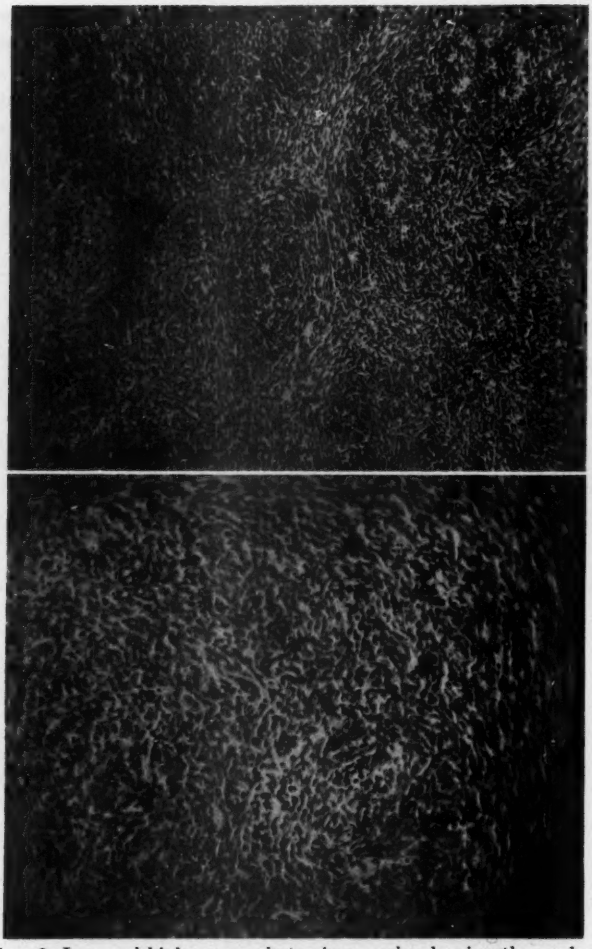

FIG. 4, Case 2. Low and high power photomicrographs showing the nuclear palisading and fasiculation characteristic of a nerve sheath neoplasm.

SUMMARY

Two hundred and seventeen mediastinal tumors of neurogenic origin have been found in a review of the literature. Only 9 of these were located in the anterior or middle mediastinum.

Two additional cases of anteriorly located neurogenic tumors of the mediastinum have been reported. One of these was a cystic lesion of the middle mediastinum and the other a solid tumor of the anterior mediastinum.

The opinions and conclusions expressed in this paper are those of the authors and are not to be construed as necessarily reflecting those of the Medical Department of the U. S. Navy, or of the Naval Service at large.

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THE FOURTH DIMENSION IN PULMONARY DISEASE

RICHARD H. OVERHOLT, M.D.* AND SHERMAN W. ATWELL*

Brookline, Mass.

Time, hailed as the *Great Healer*, also should be recognized in its competitive role of *Smouldering Menace* and *Merciless Killer*. To correlate the dimension of time with pulmonary diseases, we shall separate these disorders into three general groups, each based, not only on pathogenesis and anatomic involvement, but also on the characteristic chronological behavior.

THE INFECTIOUS DISEASES

Reversible—Unlimited (Time, The Healer)

Pneumonia and pulmonary tuberculosis may resolve spontaneously, although many cases will progress if suitable therapy is not instituted. The course of the disease is governed by two immeasurable factors: virulence of the invading organism and resistance of the host.

THE STRUCTURAL DISEASES

Irreversible—Self Limiting (Time, The Menace)

Chronic lung abscess, cystic disease and bronchiectasis represent irreversible, intrenched damage to lung structure. Although the associated inflammation may resolve, the anatomic alteration is permanent and becomes a focal point of infection capable of inaugurating new disease.

THE MALIGNANT DISEASES

Irreversible—Unlimited (Time, The Killer)

Cancer of the lung must be considered a progressive lesion. When uninterrupted, it advances by invasive stages to accomplish the death of its host.

COMMENT

Within the past decade, medical, surgical and roentgenological improvements have changed the chronological ideas of treatment for diseases of the lung. Under antimicrobial therapy, many cases of infectious disease are reversed to a state of health. In other cases, the infectious process may not be checked until a significant structural defect has been produced in the pulmonary architecture. *In malignant and structural diseases, a true cure can be effected only by surgical excision.*

With exact diagnostic information, the surgical timetable can be properly scheduled: known malignancies are excised immediately; structural defects, electively. *With the diagnosis not established by routine methods, but with knowledge of pulmonary abnormality, the only logical approach toward cure is immediate exploratory thoracotomy.*

* Overholt Thoracic Clinic, Brookline, Massachusetts.

Presented during the Louisville Assembly of The Southeastern Surgical Congress, March 9–12, 1953, Louisville, Kentucky.

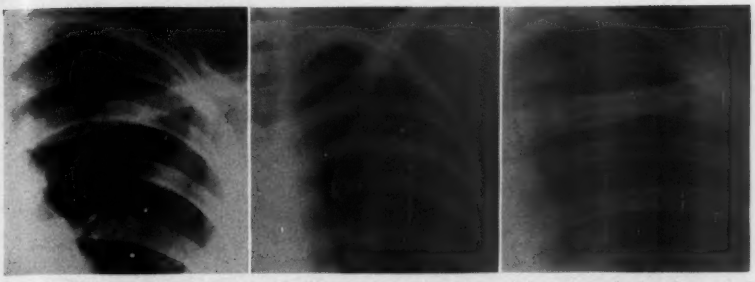

FIG. 1. First roentgenograms of three young females with tuberculosis. Case A had no symptoms, but her gastric contents contained tubercle bacilli.

FIG. 2. Effect of time: Case A refused treatment. Three months later, there were severe symptoms and roentgenographic evidence of progression. Case B refused treatment. Three months later, all evidence of an active tuberculous process had disappeared. Case C accepted treatment. When the lesion did not respond to rest and drugs, it was resected. Cases B and C are now well and negative. Case A is dead.

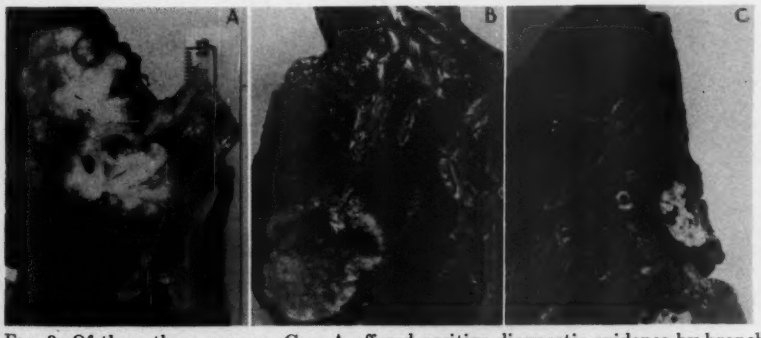

FIG. 3. Of these three cancers, Case A offered positive diagnostic evidence by bronchoscopic biopsy and Case B by cytological examination. Case C, the most curable, was negative by all preoperative tests.

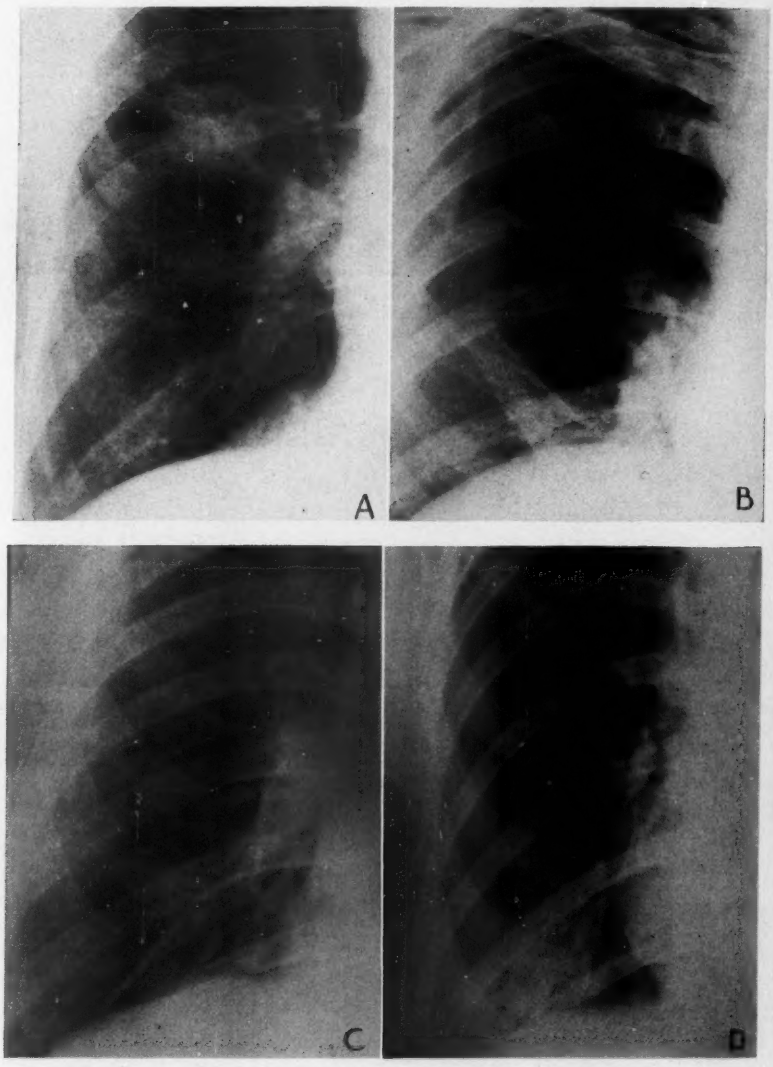

FIG. 4. The roentgenographic demonstration of bronchogenic carcinoma will usually appear as one of the above patterns. Three of these, however, are not cancer, but are (in no particular order) endobronchial tuberculosis, cystic disease and benign mediastinal tumor.

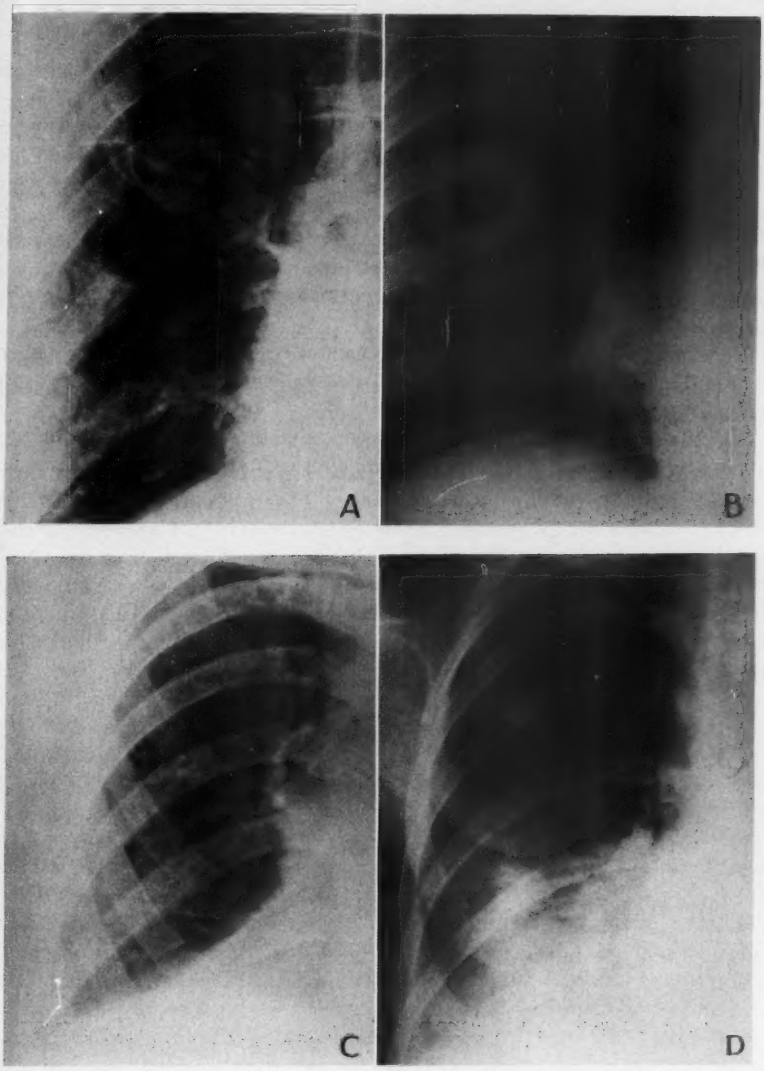

FIG. 5. The same cases as shown in figure 4 with the added dimension of time. Case A spontaneously evacuated the material from his cysts, leaving this more typical pattern. Case B was a benign thymoma. In Case C, the cancer which had acted as a valve within the bronchus grew to sufficient size to cause atelectasis of the involved segment. The endobronchial tuberculosis in case D was determined by bronchoscopic examination.

SUMMARY

Within the dimension of time, the pulmonary diseases are seldom static, and those which seem unmoving retain an insidious threat.

The infectious diseases are not static—they either progress or retrogress.

The structural diseases are irreversible since they represent permanent pulmonary damage, but progression in these diseases depends upon reinfection or mechanical abnormality.

The malignant diseases are irreversible and without any check except excision.

In the infectious diseases, the associated inflammation may recede, but sometimes not until infection has intrenched permanent structural damage into the pulmonary anatomy.

The structural and malignant diseases cannot be cured without excision.

Often, the first indication of malignant invasion is in roentgenographic demonstration of *structural* abnormality.

With the diagnosis not established by routine methods, but with knowledge of pulmonary abnormality, the only logical approach toward cure is immediate exploratory thoracotomy.

CHOLELITHIASIS AND CARDIAC DISEASE

RICHARD M. GARRETT, M.D.

Baltimore, Md.

Each of us in the course of our practice has had patients who stand out in our memory. There are 2 cases which stand out rather vividly in my memory. These were cases with gallbladder disease and associated cardiac pathology.

During the early part of World War II, I was serving as an intern in a very busy hospital. One evening I examined a patient who had right upper quadrant pain, rigidity, and a story of nausea and vomiting. He had a clinical story of intolerance to fatty food and he exhibited a mild jaundice.

It was my duty to call one of the senior attending surgeons as consultant. With so many doctors in the military service, this surgeon, like others in civilian practice, was carrying a very heavy schedule and I was reluctant to ask him to come into the hospital during the small hours of the morning unless it was imperative. In reply to his query, I stated positively that the patient had acute gallbladder disease. We prepared the patient for surgery and he was taken to the operating room. When the senior surgeon arrived at the hospital, he asked to examine the patient before operation. He found that the patient was exhibiting some mild cyanosis and was asking to have his head elevated. The operation was cancelled. The patient's cardiac condition became progressively worse and within three hours he was dead. To my embarrassment, the autopsy showed massive coronary thrombosis. But he also had a gangrenous gallbladder.

This episode was still fresh in my memory when I saw the next patient in a similar situation. On my first day in private practice, I performed a cholecystectomy on a patient with acute gallbladder disease and later found a massive coronary thrombosis. This patient survived, however, and at this time, over three years later, he is free of symptoms from both the cardiac disease and the gallbladder disease. These cases stimulated my interest in the relationship of these two disease entities.

In reviewing the literature, I find that for over half a century clinicians have insisted that there is more than an accidental association between diseases of the heart and the gallbladder. MacKenzie[2], in 1923, pointed out that the autopsy of John Hunter, who suffered from attacks of angina pectoris, showed he had coronary sclerosis and gallstones. Roberts[6], in 1929, expressed the opinion that a patient with gallbladder disease may present symptoms of angina pectoris before any characteristic gallbladder symptoms have appeared. Miller[3] in 1932 reported several cases in which gallbladder disease and coronary thrombosis were definitely associated. The incidence of both diseases simultaneously is on the increase due to better methods of diagnosis and to the fact that more people reach the age when these conditions are frequently found.

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It is then evident that the differential diagnosis of gallbladder disease and certain types of heart disease is frequently encountered. An episode of coronary occlusion or angina pectoris may mimic gallbladder disease. Error in the admitting diagnosis is frequent and may lead to surgical intervention in the patient with no gallbladder disease or may make a later cardiac cripple of a patient with no coronary disease when the patient could have been cured by surgical intervention.

The electrocardiogram is of great value but it has its limitations. Diagnostic changes may be absent if the area of myocardial damage is small, or if the leads are taken at the wrong time. Acute coronary occlusion may fail to cause electrocardiographic changes in the ensuing week or two, and may show no change at all. Acute disease of the gallbladder may be accompanied by electrocardiographic changes suggestive of myocardial infarction. Miller[3] places these cases in three clinical groups.

Group 1 consists of patients who develop acute gallbladder disease which simulates acute coronary occlusion, or vice versa. The general reaction is widespread and violent and is practically identical in the two diseases.

Group 2 includes patients who develop acute coronary occlusion and gallbladder disease almost simultaneously or within a few hours of each other.

Group 3 includes patients with chronic disease of both organs. The clinical manifestations may well serve as a reliable basis for differential diagnosis.

Differential Diagnosis

	Anginal Syndrome (Coronary thrombosis)	(Gallbladder Disease)
History:	Flatulent dyspepsia occasionally	Flatulent dyspepsia, nausea, vomiting, biliary colic.
Age:	Above 45–50 years	Any age, though common in the same age period.
Sex:	Rare in women (about 22%) 15% in my experience	Very common in women.
Pain:	Continuous, not fluctuating. Lower retro-sternal pain radiating upward to neck, and along both arms	Paroxysmal, right upper abdominal pain radiating to the right shoulder area.
Dyspnea & Cyanosis:	Present	Absent
Pulse:	Rapid, usually	Slow (due to reflex vagal stimulation) or rapid
Pulmonary congestion:	Present (if left ventricular failure)	Absent
Cardiac Signs:	Enlargement of heart, gallop rhythm, pulsus alternans, pericardial rub.	Usually absent, but both Reisman (1907) and Osler described mitral systolic murmur during biliary colic.
Blood Pressure:	Progressive fall. Transient rise, occasional.	Fall
Abdominal signs:		
Rigidity	Absent	Present
G.B. area tenderness and swelling	Absent	Present
Jaundice	Absent	May be present

Various workers have found that the diagnosis is not as easy as it appears from the above diagnostic points. This paper is not a detailed discussion of all diseases from which gallbladder disease has to be differentiated. It is presented in an effort to focus attention upon the close relationship between the anginal syndrome and cholecystitis.

Of 57 consecutive patients upon whom I have performed cholecystectomies, 18 had symptoms and findings in their histories suggestive of cardiac disease. The electrocardiogram was reported as negative in 8 of these patients and revealed cardiac pathology in 10 patients. Electrocardiographic abnormalities, which are characteristic of myocardial damage, usually undergo improvement or disappear upon removal of the diseased gallbladder. The operative risk of such interference is no greater than for other operations in the same age group.

Crittenden and Ivy[1] have studied viscero-cardiac reflexes in unanesthetized dogs with control electrocardiograms on normal dogs and icteric dogs. In normal dogs, nausea, vomiting and retching induced by injections of apomorphine resulted in cardiac irregularities including heart block, cardiac arrests, tachycardia and ventricular and auricular ectopic beats. Bile salts increase the tone of the vagus and it was found that preexisting icterus increased the cardiac irregularities which occurred with nausea, retching, vomiting, and pain due to distention of the biliary passages. The intravenous injection of atropine almost completely abolished cardiac irregularities, occurring with nausea, vomiting and retching, which were produced by apomorphine.

Owen[4] concluded that it is possible that biliary tract disease, when present for a period of time, may in some way predispose the heart to visceral influence. Ravdin[5] demonstrated in a patient, who had previously experienced attacks of sternal and epigastric pain radiating to the left arm, that with over-distention of the common duct through a T tube, there was a reproduction of pain similar to the pain which the patient had previously experienced.

Chronic gallbladder disease may mimic heart disease; and there is a growing belief that chronic gallbladder disease may initiate or aggravate heart disease. Cardiac improvement following cholecystectomy for chronic gallbladder disease in patients with myocardial disease supports this belief. Marked relief from pseudo-angina may follow removal of a diseased gallbladder in patients whose symptoms are suggestive of, but not due to, true heart disease. The following case is a good example.

CASE REPORT

Mrs. L. S., aged 52, a white housewife, was admitted to the hospital on March 10, 1952 as an emergency case with a diagnosis of acute coronary thrombosis. On admission she appeared comfortable and her color was good. She stated that she had had severe, sharp pain in the left precordium going to the shoulder and right arm, relieved only by injections given by her physician. On examination there was a slight cyanosis of the lips and nail beds. Respirations were increased, blood pressure 120/80, chest clear, and breath sounds were of fair quality. The pulse was regular in rate and rhythm. The abdomen was tense but there was no

tenderness. The liver edge was palpable. No other masses or viscera were palpable. The patient was placed in an oxygen tent and was given analgesics and respiratory stimulants. She improved rapidly after admission and was placed on a cardiac regime which consisted of complete bed rest, cardiac diet antispasmodics and analgesics. As the patient resumed activity, she complained from time to time of a dull aching pain around the zyphoid process penetrating to the shoulder blades. A cholecystogram was done on April 3, 1952 and revealed the presence of a gallstone. Because of the lack of definitely proved heart disease, it was decided that a cholecystectomy should be done. A cholecystectomy was done on April 8, 1952. She withstood the procedure well and her postoperative period was uneventful. She has been entirely free of cardiac symptoms since the removal of her gallbladder.

From the above discussion, it appears obvious that any patient with clinical diagnosis of myocardial disease should have a cholecystogram done to rule out influence from the gallbladder; and by the same token, any patient with gallbladder disease should have an electrocardiogram.

SUMMARY

Cholecystitis may simulate acute coronary occlusion.

Cholecystitis may develop simultaneously with myocardial disease.

Cholecystitis may appear in its chronic form coincident with chronic coronary disease. Distention of the biliary passages and nausea and vomiting cause cardiac irregularities. Icterus increases the susceptibility of the heart to irregularities.

Marked clinical and electrocardiographic improvement of the cardiac status follows a cholecystectomy for cholecystic disease when organic disease of the heart is also present. Patients with chronic heart disease and chronic cholecystitis should have cholecystectomies notwithstanding the obvious risk. Results of cholecystectomy are particularly good when stones are present. All patients with symptoms of cardiac disease should have a cholecystogram and all patients with symptoms of gallbladder disease should have an electrocardiogram made.

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RETROGRADE JEJUNO-GASTRIC INTUSSUSCEPTION

REVIEW OF LITERATURE AND REPORT OF A CASE

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An increasingly radical attitude over the last five decades toward extirpation of the stomach has characterized gastric surgery for peptic ulcer and carcinoma. This attitude has prevailed because the benefits accrued from radical surgical intervention have thus far outweighed the failures and complications to be found. Nonetheless, technical errors with resultant mechanical complications appeared in isolated case reports along with the successes of each new method in gastric surgery.

Simple gastroenterostomy used to be the procedure of choice for obstruction or hemorrhage from intractable peptic ulcer. Before this palliative operation gave way to more definitive procedures, Lawson and Whitener[8] were able to collect 51 cases of retrograde jejuno-gastric intussusception complicating such a relatively benign procedure. That this figure culled from the literature up to 1950 was obviously on the conservative side was evident from an earlier report by Aleman[1] who was able to collect 70 cases including those from a comprehensive review of the European literature. Even this larger series probably represents less than the actual incidence since there were no reported cases from the first gastroenterostomy in 1881 to the first reported case of jejuno- gastric intussusception by Bozzi[1] in 1914. Then too, from the time Von Steber's report in 1917 stimulated enough interest in the subject to the first attempt at compilation of cases and description of the various types of such intussusception by Shackman[12] in 1940, enough reports in both surgical[7] and radiological[4, 6] literature had appeared to show that a chronic form of retrograde jejuno- gastric intussusception compatible with the patient's doing his normal work for years, was a definite entity, at least, roentgenologically. The cases of Sibley and Chamberlin[4] and of Lenarduzzi and Bonomini[1] which had spontaneous reduction before operation lend credence to the probability that there must be instances unrecognizable on reaching medical attention due to spontaneous reduction.

Since McNamara[10] published his report on retrograde jejuno-gastric intussusception following *partial gastrectomy*, there have not been more than 4 other cases (Grimes, Aleman, Vink and Toeuf) in currently available literature of such a complication of partial gastrectomy. The available Russian literature has only two reports on the subject.[5, 14] In so far as can be ascertained our case report is the first reported retrograde jejuno-gastric intussusception following partial gastric resection for *gastric carcinoma*. We may expect more frequent reports of this complication as the indications[3] for partial gastrectomy are widened.

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CASE REPORT

C. B., aged 64, a colored man, was admitted to the Jefferson Davis Hospital with the chief complaint of nausea and vomiting for the last year. A 30 pound weight loss and anorexia were also noted by the patient in the same period of time. The preoperative diagnosis was carcinoma of the stomach and at operation on Jan. 31, 1944, a prepyloric ulcer of the malignant type was found with no gross involvement of any of the regional lymph nodes. An incidental finding was cholelithiasis. A partial gastrectomy, removing the pyloric half of the stomach, was done and a Polya gastro-jejunostomy accomplished. The patient had an uneventful post-operative course and was dismissed on the twelth day after operation.

The pathologic diagnosis was adenocarcinoma of the stomach, Grade II. Comment was made by the pathologist as to the unfavorable prognosis because neoplastic tissue was found

FIG. 1. The cut edge on the left half of the photograph represents the line of transection of the stomach in its middle third during the original partial gastrectomy for ulcerating prepyloric carcinoma performed Jan. 31, 1944. The mucosal side is at the top of the photograph. The extensive infiltration of the growth down to the muscle layer as well as to the proximity of carcinoma cells to the line of resection is well shown.

within the muscularis in the section taken close to the line of amputation of the stomach. Figure 1 is a reproduction of the original slide from the carcinomatous ulcer near the line of ablation.

Beginning a few weeks after operation and for the next eight years, the patient was subject to recurrent epigastric pain, postprandial nausea and vomiting and general inability to tolerate heavy meals. No bleeding was noted at any time but there was a gradual weight loss reaching 25 pounds during the entire period.

The patient was readmitted Nov. 9, 1952 with the history that the above-mentioned symptoms became more acute and in addition, massive regurgitation of coffee-ground vomitus was observed two weeks prior to the second admission. The patient was quite dehydrated on admission. The blood pressure was 130/70 mm Hg., pulse was 100 per minute and respirations were 18 per minute. The examination of the head, neck and chest was

essentially negative. The upper abdomen was moderately distended with slight muscle splinting. There was an old upper right paramedian incisional scar which was well healed. Tenderness was minimal and peristalsis slightly hyperactive. Rectal digital examination was negative with scanty brown feces obtained from the ampulla. The hemoglobin determination on admission was 13 Gm. per 100 cc. and the urinalysis showed slight traces of albumin and occasional red and white blood cells. The feces was slightly positive for occult blood. The vomitus was strongly positive for occult blood. Serum sodium was 134.9 mEq. per liter, chlorides were 105 mEq. per liter, CO_2, 29 mEq. per liter and blood urea nitrogen was 28 mg. per 100 cc.

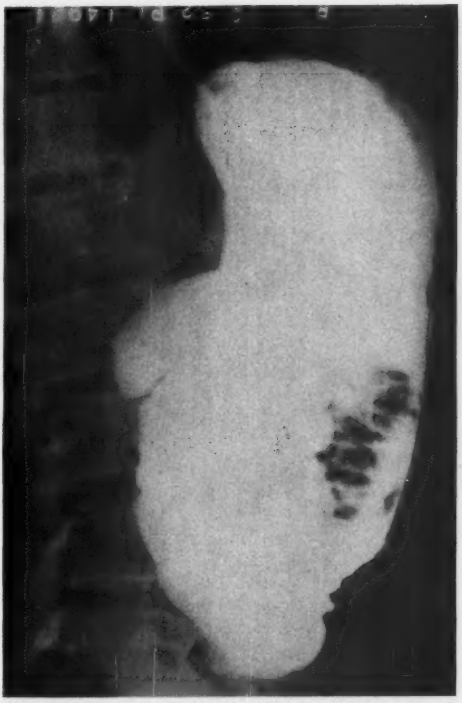

FIG. 2. The valvulae conniventes (Kerkring folds) are represented by the radio-translucent area with the stepladder-like effect hugging the greater curvature side of the stomach. The hypertrophy and dilatation the gastric stump had undergone during the eight year lull between the two operations is shown. The tongue-like projection from the lesser curvature side is the afferent limb of the jejunal loop, shown filled with the contrast medium.

Over a period of three days the patient was hydrated, decompressed with a nasogastric tube and an upper gastro intestinal series was made. Figures 2 and 3 show the roentgenograms of our case with the typical configuration of Kerkring's folds within the stomach and the absence of the contrast medium beyond the efferent jejunal limb.[6] These roentgenologic findings were unrecognized prior to operation. The preoperative diagnosis was upper small intestinal obstruction, etiology undetermined.

Surgery, performed on the fourth day after admission, disclosed a retrograde jejunogastric intussusception involving 15 inches of thickened jejunum. The intussusception began 12 inches from the ligament of Trietz and the head of the intussusceptum projected

2 inches proximal to the gastro-jejunostomy stoma which was 2 inches wide. There was no gross evidence of any tumor. The involved jejunal segment was viable and the intussusceptum was reduced, the abdomen closed and the patient was sent back to the ward in good condition.

Figure 4 is a sketch of the operative findings. The pathology in the retrocolic gastro-jejunostomy anastomosis has been clarified by omitting the overlying transverse colon and mesocolon.

The post-operative course was uneventful. The patient was tolerating a full diet on the fourth day. The sutures were removed and the patient was dismissed on the seventh day

FIG. 3. This figure shows the gastro-jejunostomy site with the *streaming* pattern of the valvulae conniventes in the jejunal loop strongly suggesting the retrograde invagination of the loop through the stoma.

after operation. He was seen one month after the operation with no complaints except dragging pain referable to a direct inguinal hernia.

Relationship to original surgery. From a cancer survival standpoint, this patient represents an 8 year *cure* of carcinoma of the stomach verified by re-exploration of the abdomen.

To our knowledge, this is the first reported case of retrograde jejuno-gastric intussusception following gastric surgery for carcinoma. In the 70 cases reported by Aleman, all but 1 followed gastric surgery for peptic ulcer. The exception was

a case of gastroptosis. None of his compiled cases followed gastric resection for carcinoma, a fact which he attributed to the failure of such patients to survive long enough for the condition to develop. As pointed out by Perman and Aleman,[1] the condition of jejuno-gastric intussusception is usually a late complication coming on several years after initial surgery.

Mortality rate. The mortality rate for the acute severe type is as high as 10 per cent[1] for cases where operation is undertaken even as early as 48 hours after the onset of symptoms. The mortality rises to 35 per cent[11] and even up to 50 per cent or more[1] for delayed operations.

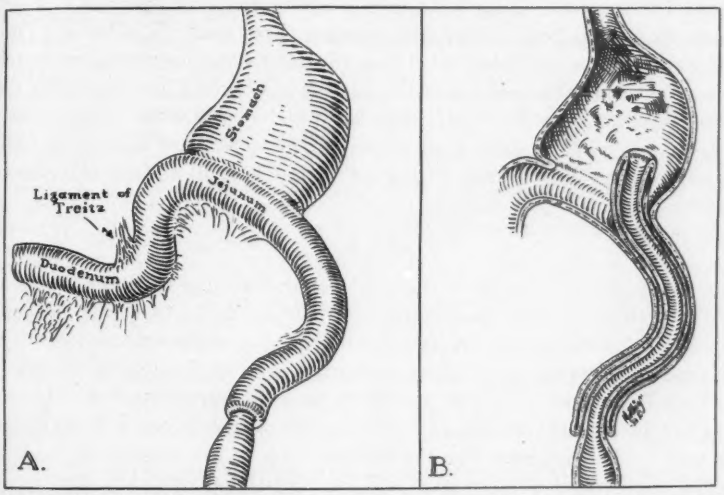

FIG. 4. A. In this sketch the pathology as found at operation is amply demonstrated. The transverse colon and mesocolon as well as other adjacent viscera have been purposely omitted for clarity.

B. A longitudinal section of the sketch is presented to illustrate the relationship of the intussusceptum to the stoma and the mechanism of complete obstruction of the efferent jejunal limb.

N.B. The dilatation of the afferent limb as well as that of the gastric stump which was not as apparent at operation as one would have expected from the completeness of the obstruction was relieved effectively by preoperative nasogastric suction.

DIAGNOSIS

Diagnosis then must be made early.

McNamara,[10] Aleman,[1] Chamberlin[4] and other investigators recognized the existence of the relatively benign chronic recurrent form sometimes demonstrable only by roentgenogram and absent upon the operating table. Our case represents an example of the chronic recurrent form, seen during an acute episode.

It may be stated categorically that in any patient with a previous history of gastroenterostomy or with any form of gastric surgery patterned after the Billroth II procedure, whose complaints subsequently are referable to upper small

intestinal obstruction, the differential diagnosis must include retrograde jejuno-gastric intussusception. The index of suspicion should increase if, notwithstanding obvious obstruction, plain roentgenograms of the abdomen fail to reveal obstructed small bowel loops. This condition demands emergency contrast studies of the upper gastrointestinal tract if attempts to reach an early correct preoperative diagnosis are to succeed.

About 85 per cent[1] of the acute severe types will show pathognomonic roentgenologic evidence consisting of recognizable Kerkring[1] folds (valvulae conniventes) within the stomach displacing the contrast medium away from the stomal site. In 18 cases collected by Aleman with roentgenologic examinations, 17 showed this typical filling defect but 3 were wrongly interpreted.

Failure to demonstrate these folds, occurs in those cases where the apex of the intussusceptum has not invaginated past the gastroenterostomy stoma or where spontaneous or barium meal induced reduction has taken place during the roentgenologic examination. In itself, high intestinal obstruction with or without roentgenologic corroboration is an indication for emergency surgery so this 15 per cent roentgenologic failure should not be a deterrent to improving the salvage rate.

Etiology

The much higher incidence of the condition in simple gastroenterostomy compared with that following gastric resection (a ratio of 13 to 1) has led original reviewers[1] to assume that hydrochloric acid irritation of the anastomosed jejunal loop, producing spasm and violent peristalsis, is a definite etiologic factor. Aleman's excellent review of the subject offers the interesting hypothesis that intussusception eventually proceeds in the retrograde manner because of the tendency for a more mobile segment like the efferent limb of the jejunum to invaginate into the more fixed gastric stump. This holds true especially for retrocolic anastomoses.

An additional explanation[1] for the rarity of retrograde jejuno-gastric intussusception following partial gastrectomy compared with its incidence in simple gastroenterostomy is the fact that counterpressure against an invagination within the gastric stump following partial gastrectomy becomes greater than when all the stomach is still present as is the case in simple gastroenterostomy. Consequently, the tendency to retrograde invagination past the stoma is minimized and if it occurs, spontaneous reduction is more apt to occur.

This leads to one strong inference: the smaller the gastric stump left after partial gastrectomy, the less the chances for the development of retrograde jejuno-gastric intussusception. Should our patient come back later with recurrence of his intussusception symptoms, a possibility quite likely considering his history after his gastric resection, further resection of the gastic stump would seem to be indicated

SUMMARY

The first case of retrograde jejuno-gastric intussusception in a patient with previous partial gastric resection for gastric carcinoma is reported. The patient

has survived the initial cancer surgery for eight years and has survived the reduction of his intussusception with no further recurrence of symptoms to date.

A review of the literature with the addition of a case is presented in an effort to increase awareness of the condition in the differential diagnosis of high intestinal obstruction particularly when previous gastrojejunostomy had been done.

The treatment of the condition is reduction of the intussusception. Should the condition of the patient permit, it is probably advisable to do a more adequate gastric resection in an attempt to prevent recurrence of the intussusception.

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REPAIR OF THE NEGLECTED RUPTURED AND SEVERED ACHILLES TENDON

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The Achilles tendon is the strongest tendon in the body. It narrows and becomes thickened as it descends and again spreads out to be inserted into the middle part of the posterior surface of the os calcis. Complete rupture of this tendon is a relatively rare condition. Partial rupture probably occurs more frequently than realized and usually responds to conservative measures. On the other hand, a partially ruptured Achilles tendon may progress to a complete rupture.

Etiologically, ruptures of the Achilles tendon are of three types: (1) direct, resulting from a severe trauma over the tendon; (2) indirect, resulting from a fall or from athletic activities, such as volleyball, basketball, or high jumping, where the individual lands on the forefoot with the ankle in flexion and the knee in extension; and (3) pathological, due to atrophy of the muscle and tendon, adhesions about the tendon or from a generalized condition such as tabes. The patient presents a clear-cut clinical picture. There is inability to *take off* on the involved extremity. Immediately there is a hematoma over the site of the rupture, but this usually subsides in a few days. The patient may be able to flex his foot with the long flexors of the toes. The diagnosis should be made immediately and surgical repair of the ruptured tendon should be made before atrophy and adhesions develop. Henry[2] described the appearance of the tendon at operation. The proximal tendon fragment and muscle is retracted upward and the tendon ends are frayed and fibrillated. The Achilles tendon may rupture at (1) the musculotendinous juncture, (2) the mid-tendinous region, and (3) at the insertion into the os calcis.

The diagnosis is usually more clear-cut in the completely severed cases with laceration over the tendon; but the extent of the injury is not always realized, and the skin alone may be sutured. Any laceration over the tendon should be adequately explored to evaluate tendon function. The Achilles tendon may be only partially severed and some time later the tendon fragments may completely separate.

This paper is concerned primarily with the neglected cases of rupture and severance of the Achilles tendon.

From a historical viewpoint, Keith[3] stated that John Hunter, in 1768, at the age of 40 years, is reported to have ruptured his Achilles tendon while dancing. He treated himself by encircling his calf with a bandage to prevent involuntary movements and applied an apparatus to protect the muscles during the night.

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He raised his heel and apparently a full union of the tendon ends occurred. Hunter became interested in the healing of tendons. He cut the tendon of a deer to, (1) prevent it from jumping over a fence, and (2) to study the method by which a tendon would heal. He found that a space between the severed ends of a tendon did not prevent union. Coagulated lymph was formed between the ends which could eventually provide a strong bridge over a wide gap. After Hunter's death it was found that the cicatricial area in his own Achilles tendon had become calcified. It is the opinion of the author that Hunter probably sustained a partial rupture of his Achilles tendon, since the treatment given himself would hardly have been adequate to bring about a spontaneous healing of the tendon ends had he sustained a complete rupture of his Achilles tendon. The observation of Hunter in 1768 that the gap between the ends of the tendon was *bridged* with scar tissue is substantiated by the gross findings at time of operation, as described later in this paper.

Various methods of repair of old, untreated ruptures of the Achilles tendon have been described. Zadek[7] suggested the use of three strips of fascia lata, each ½ inch wide and 6 inches long. These strips were woven into the proximal and distal tendon fragments and then sutured to each other. Turning down two strips of fascia from the superficial surface of the calf muscles is another method of repair. Toygar[6] mentioned that *the elimination of distraction* is the major factor in treatment. He advocated forming a proximal-medial and a distal-lateral strip from the tendon ends, pulling the proximal strip down and the distal strip up, and leaving both attached to their respective tendon ends. He believed that by this method the circulation of the tendon strips was not damaged. An advantage in this method is the use of a single incision. A free tendon graft may be considered, and Platt[5] recommended the transplantation of the peroneus longus and tibialis posterior tendons into the os calcis. The plantaris tendon may be utilized by leaving it attached to the os calcis and sectioning it high in the calf. The Achilles tendon may be divided in a "Z" manner and shortened.[2] Tension from retraction of the proximal fragment may be minimized by the removable wire staple suture of McLaughlin.[4]

Another method of using fascia lata in the repair of old, neglected, ruptured and severed Achilles tendons is here suggested.

Operative technic: With the patient under general or spinal anesthesia and lying prone on the table, the entire involved extremity is prepared from the gluteal fold down to and including the foot. A curved incision from 4 to 6 inches long is made lateral to the Achilles tendon. It is desirable to avoid a scar over the tendon. A medial incision is rather hazardous because of possible injury to the posterior tibial vessels and nerve. The skin is retracted medially and the area of involvement is easily exposed. The ruptured or severed site is usually found to be replaced with fibrous tissue. A normal Achilles tendon has a smooth glistening appearance and is not adherent to the surrounding soft tissues. An old ruptured or severed Achilles tendon is dull in appearance, irregular, and is adherent to the soft structures (fig. 1). By further dissection of the scarred sheath the distal fragment attached to the os calcis is exposed (fig. 2). The proximal tendon frag-

ment is found to have retracted. The intervening fibrous tissue is excised leaving a gap of varying degree between the tendon ends. A purse string suture is inserted into each tendon end to hold the tendon fibers together. At this time, the amount of fascia lata needed in the repair can be readily determined.

The leg wound is covered with a sterile towel and a long incision is made over the lateral aspect of the thigh exposing the fascia. A large rectangular piece of fascia lata, the size depending upon the defect in the Achilles tendon, but usually about 4 by 12 centimeters, is removed (fig. 3). To save operating time, this part

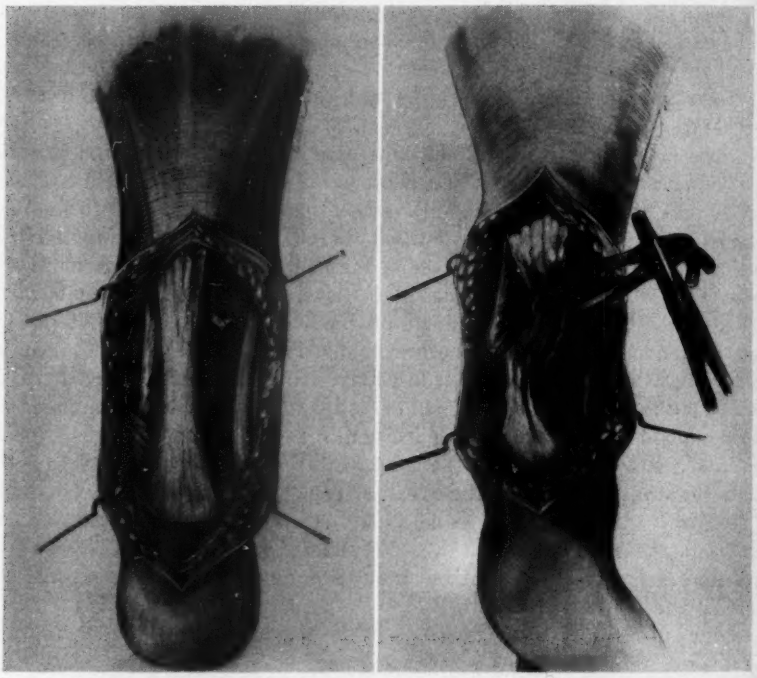

FIG. 1. Schematic drawings of a normal and old ruptured Achilles tendon. Note smooth contour of the normal compared with the irregular, relaxed Achilles tendon. There is no clearcut lack of continuity as the ruptured area is replaced with scar tissue.

of the operation may be done by another surgeon. Removal of a large piece of fascia lata apparently does not have any untoward effects, and herniation of the muscle does not occur as it frequently does when a small strip of fascia lata is removed.

The rectangular piece of fascia lata is rolled on itself as one would roll a cigarette, and a fine suture is inserted to keep the roll intact (fig. 3). Holes are made with a clamp through both tendon ends, and the roll of fascia is inserted. The posterior part of the fascia is unrolled and narrow strips about $\frac{3}{8}$ of an inch wide are separated from, but left attached to, the fascia itself. The fascia is overlapped

and sutured with interrupted sutures or, preferably, with a pull-out stainless steel wire. The strips are threaded back and forth through the tendon ends to further strengthen the repair (fig. 4). Very fine stainless steel sutures may be used to attach the fascial flaps to themselves and to the tendon ends. As the blood

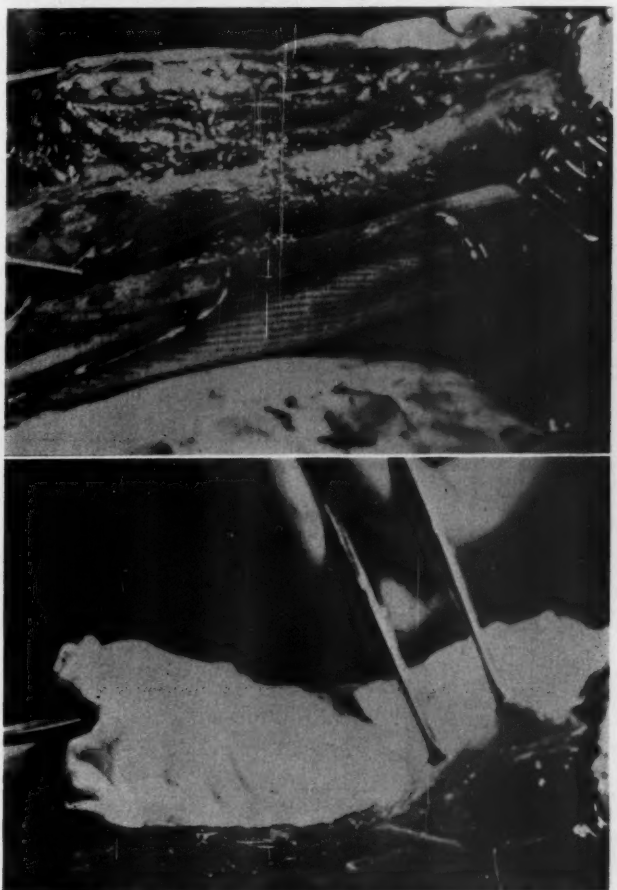

FIG. 2. (Top) Photograph of old ruptured Achilles tendon. The area of rupture has been replaced by scar tissue. The distal portion of the tendon has retracted downward and appears as a bulbous swelling. (Bottom) The scarred sheath has been opened and the distal tendon stump can be seen.

supply in this area is not good, there is danger in the use of too much silk. A tourniquet is not necessary.

Postoperative care: A long-leg cast is applied from the toes to the groin with the knee and ankle flexed about 35 degrees. This is important. The cast may be changed in several weeks, the sutures removed, and another cast applied with

the knee and ankle in less flexion. Depending on the integrity of the fascial repair, the cast is worn for six to eight weeks. A light brace may be applied to prevent forced, sudden dorsiflexion of the foot. The heel may be raised to maintain plantar flexion. Physical therapy is instituted following the removal of the cast to restore motions which were lost by cast immobilization. Full return of motion

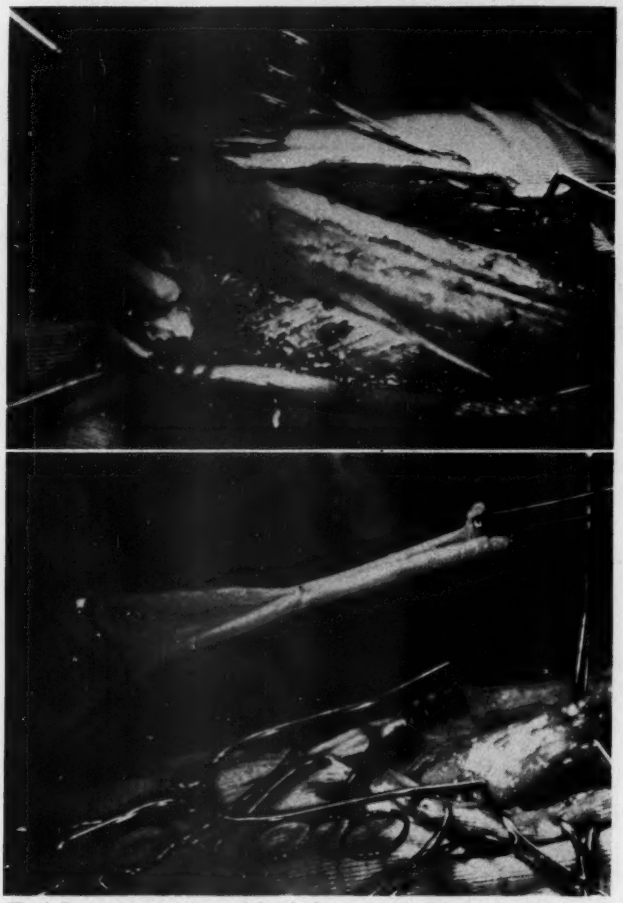

FIG. 3. (Top) Rectangular piece of fascia lata removed from the thigh. (Bottom) The fascia lata is rolled and a single suture is placed in the center to maintain the position of the rolled fascia.

of the knee and ankle may be expected. The following cases present typical histories and findings:

Case 1. W. O., a man, aged 30, sustained a rupture of the left Achilles tendon while playing basketball. Fascia lata repair was made four months later. In three months he was able to bear his entire weight of 200 pounds on his left forefoot.

Case 2. D. J., a man, aged 55, felt a *snap* in the posterior aspect of his right ankle while playing volleyball. There was immediate inability to stand on the ball of the right foot, and loss of normal contour of the Achilles tendon and a limp (fig. 6). He was unable to plantar flex the right ankle against the examiner's hand. Nine weeks later, a fascia lata repair

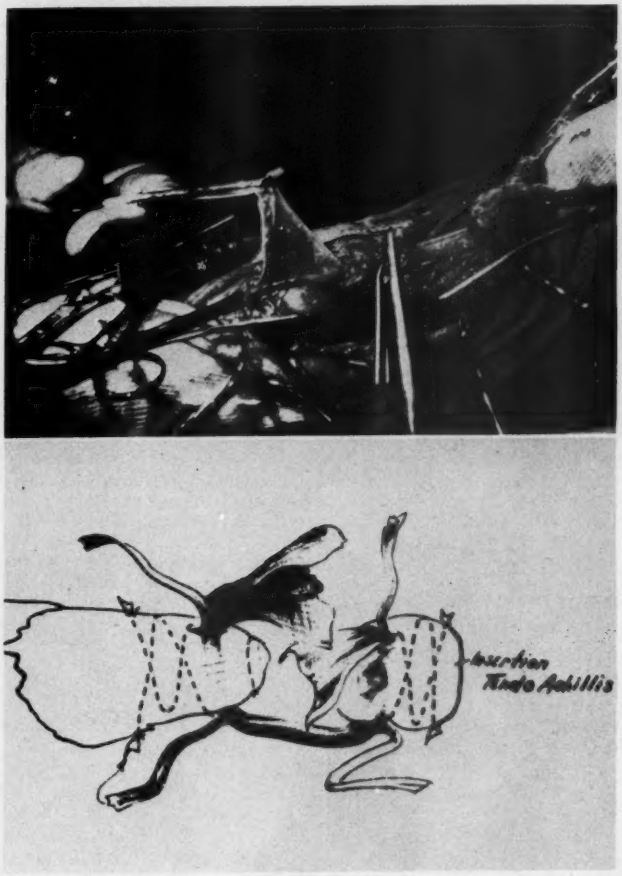

FIG. 4. (Top) The rolled fascia has been passed through the proximal and distal tendon ends after removal of the intervening scar tissue. (Bottom) Schematic drawing showing the purse string sutures in the tendon ends and the manner in which the strips of fascia are interwoven in the tendon ends.

was made. Eight weeks postoperatively he was able to raise both heels from the floor and actively plantar flex the right ankle against the resistance of the examiner's hand.

Case 3. S. M., a woman, aged 19, sustained a laceration over the left Achilles tendon. There was an initial suture of the skin, but a diagnosis of a severed tendon was not made at that time. She walked with a limp and was unable to raise the left heel from the floor. A fascia lata repair was made seven months later. Ten weeks postoperatively she was able to bear her entire weight on the left forefoot.

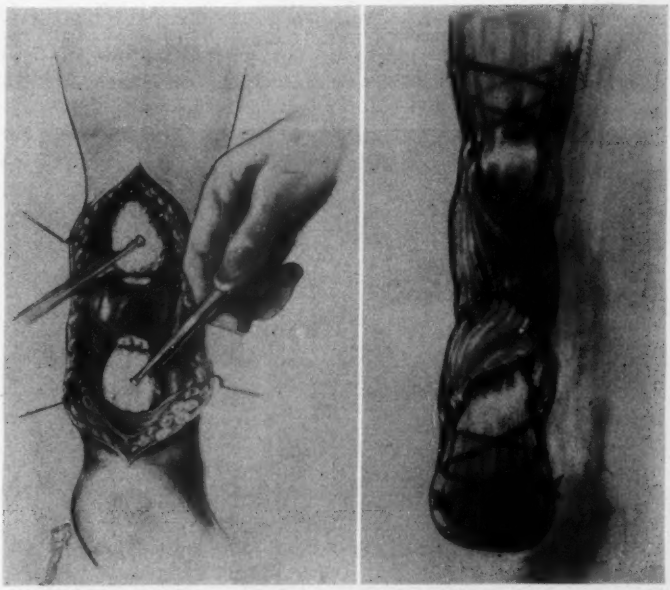

FIG. 5. (Left) Showing the space between the tendon ends after the scar tissue has been removed. (Right) Sketch showing the completed operation. Fewer sutures are required than shown. Pull-out stainless steel sutures may be used instead of silk sutures.

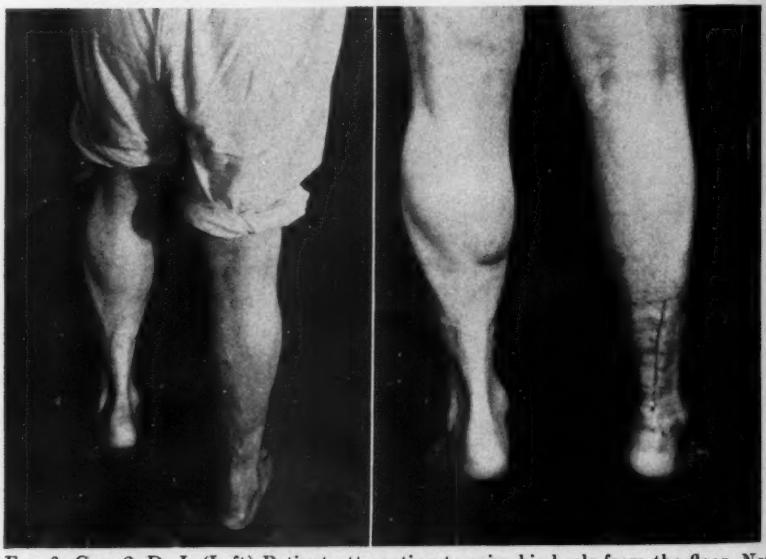

FIG. 6. Case 2. D. J. (Left) Patient attempting to raise his heels from the floor. Note normal contour of the left Achilles tendon and flattening on the right side. The right heel cannot be raised from the floor. (Right) Able to raise right heel from the floor eight weeks postoperative.

Case 4. P. D., a woman, aged 37, sustained a laceration over the left Achilles tendon. The laceration of the skin was sutured. Since the injury she had been unable to stand on her left forefoot and walked with a limp. Examination revealed loss of contour of the Achilles tendon. The patient could easily stand on her right forefoot but could not raise her left heel from the floor. Lateral roentgenogram of the normal ankle, with soft tissue technic, showed the normal triangular area of lessened density between the Achilles tendon and the deep flexor tendons anteriorly. A similar exposure of the involved ankle showed a loss of this area of lessened density due to scar tissue formation in and about the tendon ends. A fascia lata repair was made four weeks after injury. She obtained an excellent result and eight weeks postoperatively she could raise her heel from the floor and bear her weight on the metatarsal region of her foot (fig. 7).

Case 5. F. Z., man, aged 34, sustained a rupture of the right Achilles tendon while playing volleyball. There was a sudden sharp pain over the right Achilles tendon and inability to

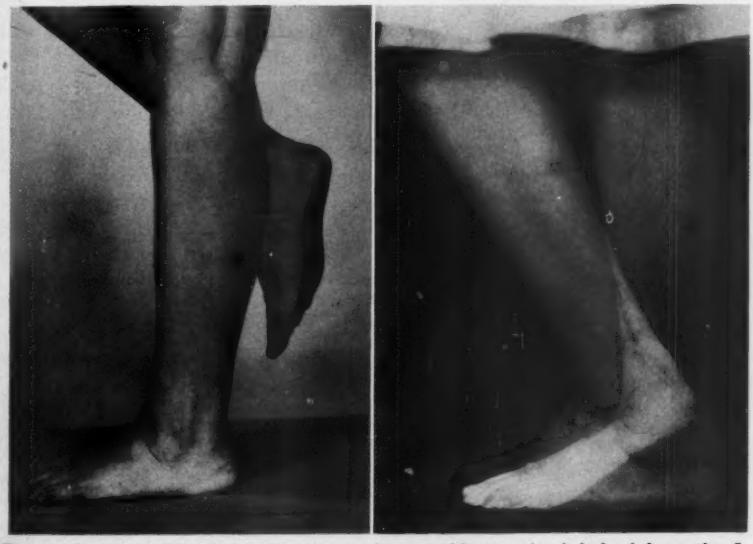

FIG. 7. Case 4. P. D. (Left) Showing patient unable to raise left heel from the floor. Note defect in contour of tendon. (Right) Double exposure photograph showing patient able to raise left heel from the floor eight weeks postoperative.

take off on his right foot. He was able to bear full weight on his left forefoot but was unable to raise the left heel from the floor. A fascia lata repair was made three months following the initial injury.

Case 6. E. G., man, aged 38, struck the Achilles tendon forcibly against the rung of a chair. A fascia lata repair was made three weeks later.

Case 7. R. H., woman, aged 57, sustained a laceration over the left Achilles tendon. The skin was sutured, but a diagnosis of injury to the tendon was not made at the time. She had difficulty in walking and inability to stand on the left forefoot. A fascia lata repair was made six months later.

Complications: The only complication in this series was a delay in healing of part of the incision in 2 of the patients.

Results: The results were uniformly good, and the patients were all satisfied with their improved gait and *take off*.

Of the 7 cases briefly presented, it is interesting to note that the three female patients sustained lacerations over the Achilles tendon. Apparently the extent of the injuries were not appreciated at the time of the suture of the lacerations. The 4 male patients all sustained ruptures of the Achilles tendon, 3 from indirect trauma, and 1 from direct trauma over the tendon.

SUMMARY

A method of using fascia lata in the repair of old neglected, ruptured and severed Achilles tendons is suggested.

The gross pathological findings at operation are described.

Tightening up the *lengthened* tendon is the essential factor in the operation.

The operative repair suggested is believed to give considerable stability to the Achilles tendon.

No untoward effects were noted when a large rectangular piece of fascia lata was removed from the lateral aspect of the thigh.

Seven additional cases of this relatively rare injury are reported.

Note: The author wishes to express his appreciation to Dr. J. William Littler and Mr. Kucera for the drawings used in the illustrations.

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RECURRENT CARCINOMA OF THE AMPULLA OF VATER

A CASE REPORT

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The opportunity of successfully resecting a recurrence of an ampullary carcinoma following previous transduodenal excision is rare. Booher and Pack[1], in a recent report, reviewed four such cases and added one of their own. The case here presented demonstrates again that transduodenal resection for cancer of the ampulla is an inadequate operation and that pancreaticoduodenal resection is necessary to achieve favorable results with this apparently limited growth. The recurrence of the carcinoma in this case began at the limits of the previous transduodenal resection, and thus shows with remarkable clarity the major shortcoming of transduodenal resection for ampullary carcinoma. It may appear redundant or repetitious to emphasize the point, but it is from the group of periampullary carcinomas that the greatest number of five year survivals occur when radical pancreaticoduodenal resection is employed as the method of treatment.

CASE REPORT

A. R. S., a 71 year old white man, was first admitted to the Macon Hospital on Oct. 22 1950 with a history of malaise of three months' duration and of progressive jaundice with clay-colored stools of six weeks' duration. Weight loss was marked. For several days before admission the stools had been greenish-black in color. The past history and family history were negative. On physical examination the patient was cachectic and deeply jaundiced. Except for a palpable liver edge nothing of note was discovered. The hemoglobin was 23 per cent with 1,050,000 red blood cells per cu. mm. The urine and stool were negative for urobilinogen but the stool was positive for occult blood. Roentgenologic studies of the colon and gastrointestinal tract were considered normal. Transfusions to a total of 3000 cc. were given preoperatively and the patient was explored with the preoperative diagnosis of carcinoma of the ampulla of Vater.

At operation the common duct was found to be enormously dilated. Through the wall of the duodenum a small mass could be palpated. The duodenum was opened and a reddish tumor 1½ cm. in diameter was found to occupy the area of the ampulla of Vater. This tumor was resected locally and the cut ends of the common bile duct and pancreatic ducts were anastomosed to the duodenum. A short section of rubber tubing was sutured into the pancreatic duct. The common bile duct was then opened and the long arm of a T tube was passed into the duodenum. After closure of the common duct and duodenum, a contracted gallbladder was excised. The postoperative course was satisfactory. He was discharged on the thirteenth postoperative day. The T tube was removed two months after operation.

The pathologic report was as follows:

"Resected duodenum containing papilla of Vater. A small segment of the common duct is included and follows directly through the midportion of an

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elongated tumor nodule 1½ cm. in greatest diameter. The lips of the papilla are deeply congested and edematous, exhibiting central ulceration."

"*Microscopic section* (fig. 1.) taken through the ampulla shows mucus producing adenocarcinoma originating from the epithelium of the ampulla of Vater. The malignancy freely invades the large muscle bundles of the ampullary sphincter. *The tumor appears to be completely excised.*"

This patient remained well until June 1951, at which time, notwithstanding a complaint of general fatigue, examination was negative. In the following month an ill-defined mass could be palpated beneath the right subcostal scar. By September 1951, the mass was obvious, with attachment to the scar and with recurrence in the scar. Tissue biopsy of a nodule presenting in the scar was reported

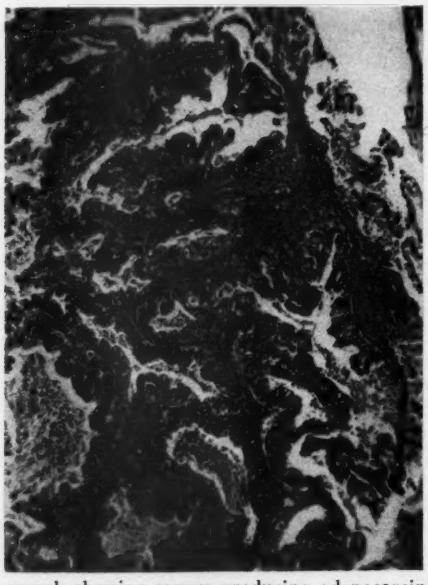

FIG. 1. Photomicrograph showing mucus producing adenocarcinoma originating from epithelium of ampulla of Vater.

as adenocarcinoma. A gastrointestinal roentgenologic study was not diagnostic of duodenal involvement. The patient was then readmitted to the hospital.

On admission to the hospital on Oct. 12, 1951, the patient complained of weakness and excessive fatigue during the previous three months, and stated that he had lost eleven pounds of weight since early summer. Jaundice had not recurred and the stools had been of normal color.

Examination revealed a slender white man, 72 years of age. Recent weight loss was evident. No jaundice was present. Physical examination was negative except for the presence of a mass attached to and extending beneath the right subcostal scar. The scar itself was not involved by tumor. A chest roentgenogram was normal. Admission blood work showed 13.9 Gm. of hemoglobin with 4,200,-

000 red blood cells per cu. mm. The urine was normal. He was operated upon the day following admission.

Under endotracheal anesthesia an elliptical incision was made around the old scar and tumor area. Medially this incision was carried into the peritoneal cavity. The mass appeared to arise from the area of the duodenum and was movable laterally. Superiorly the muscles were freed from the costal margin, and the edge of the right lobe of the liver, which was attached to the mass, was cut across. The mass was then peeled away from the liver and gallbladder bed. The hepatic flexure of the colon was adherent to the mass but grossly not involved. Adhesions were severed and the duodenum was mobilized. Dissection posterior

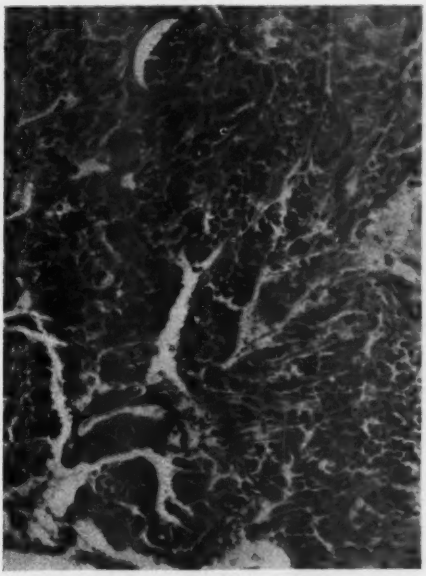

FIG. 2. Photomicrograph showing an anaplastic mucus producing columnar cell adenocarcinoma exhibiting a somewhat greater differentiation than the original growth examined one year ago as shown in fig. 1.

to the duodenum and the head of the pancreas revezled that the mass did not extend retroperitoneally. Pancreaticoduodenal resection was then done. The cut end of the pancreas was closed and sutured against the side of the jejunum. Several large rubber dam drains were placed to this area and brought out through the lateral margin of the wound. The abdomen was closed in layers using interrupted sutures of no. 00 cotton. The operating time was three and one-half hours.

The pathological report of the pancreaticoduodenal resection was as follows: "The specimen is composed of skin, abdominal wall, attached intestine, liver, and pancreas. The mucosal surface of the ampulla is free of tumor; however, one centimeter cephalad, the duodenum is ulcerated and grossly involved by tumor. The distal resected portion of the common duct is surrounded by yellowish-gray

tumor. The attached abdominal wall and skin are infiltrated by an extension of the growth. The pancreatic and liver tissues are not grossly involved. The tumor is apparently completely excised, as the resected margins of the specimen show no gross evidence of tumor invasion."

Microscopic: Examination shows an anaplastic mucus-producing columnar cell adenocarcinoma exhibiting a somewhat greater differentiation than the original growth examined one year ago (fig. 2.).

A pancreatic fistula of major proportions developed postoperatively. Constant suction was employed to combat this, and the wound edges and skin were protected by using a beef extract-hydrochloric acid paste. On the seventh postoperative day feces drained from the fistula tract. These fistulas presented a major postoperative problem. However, electrolyte balance was maintained and no intra-abdominal infection occurred. The patient was discharged on Jan. 10, 1952, approximately three months after operation. At this time his abdominal wound was healed and he was in apparent good health. Two days later he died suddenly at home with signs of a typical coronary occlusion. No autopsy was obtained.

DISCUSSION

The initial operation upon this patient was undertaken with full realization of the inadequacy of local excision of carcinoma of the ampulla of Vater. It was believed that this relatively simple procedure was consistent with the patient's ability to withstand major surgery. This was a decision in which the present surgeon (J. P. W.) concurred. Succeeding events demonstrated that it would have been wiser to do a two-stage pancreaticoduodenal resection than to temporize in the manner selected. Cattell and Pyrtek[2] have emphasized this point. Lhey reserve the two-stage operation for just such poor risk patients as this one.

The resected specimen obtained at the second operation demonstrates adequately the deficiencies of the previous transduodenal excision of the carcinoma. At the first operation all visible tumor was widely removed and in the specimen resulting from the pancreaticoduodenal resection the new ampulla was free of tumor. However, 1 cm. cephalad from the new ampulla and duodenum showed recurrent growth. The common duct, just proximal to its duodenal stoma, was solidly surrounded by tumor. The recurrence grew upward along the drainage tracts to the body surface, suggesting that the operative field had been contaminated with viable cancer cells during the transduodenal resection, and also that possibly viable cancer cells had remained within either the duodenum, the common duct, or the pancreatic ducts to flourish and grow with the passage of time. The location of these recurrences indicates the danger of local resection for ampullary cancer as well as demonstrates that the manipulations during the operative procedure undoubtedly serve to *seed* the area with cancer cells.

Pathologic examination of the specimen following the initial operation indicated a complete excision of the tumor. The hope engendered by this report was not sustained, since the tumor rapidly recurred. Miller, et al[3] have shown that in 10 (79.9 per cent) of 13 instances in which transduodenal excision was done, malignant cells were noted at the periphery of the sectioned specimen. Only 1 of

these 13 patients survived for three years, and he had recurrence of the cancer. When one recalls that the primary lesion and its zone of spread should be removed if cure is sought, transduodenal resection of periampullary carcinoma appears more in the light of removal of tumor for biopsy than a definitive cancer operation directed toward cure.

It is well recorded that the results obtained by pancreaticoduodenal resection of carcinoma of the periampullary region and the head of the pancreas are not encouraging. The operation, notwithstanding its fascination for the surgeon attempting to meet the technical difficulties produced by the position of these cancers, remains a more local cancer operation than those possible for the breast, stomach, colon, and extremities. Orr[4] has recently recorded from the literature 17 five or more year survivals, and it is well to point out that of the 15 in which the location of the lesion was mentioned, 11 were of the papilla or ampulla of Vater. This would indicate the usefulness of pancreaticoduodenal resection for these lesions.

It is beyond the scope of this case report to review the many discussions and papers concerning the treatment of cancer of the periampullary region and the head of the pancreas, but for surgeons it would be sufficient to repeat that cancer of this area is not cured by hope, but by bold, radical surgery, and that acceptable palliation is achieved more often by radical surgical resection than by the various operations which by-pass the lesion.

SUMMARY

In the case reported here, treated by transduodenal excision of an ampullary carcinoma, rapid recurrence developed notwithstanding the microscopic findings of complete removal of the tumor. It was unusual that the tumor recurrences could be removed by pancreaticoduodenal resection one year following the primary operation. Such is not the usual course of events.

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THE MANAGEMENT OF POSTOPERATIVE PULMONARY BRONCHIAL AND VASCULAR CONGESTION

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Accumulation of bronchial secretions with resulting atelectasis and major or minor degrees of anoxia still constitutes a serious problem in the postoperative patient, especially the elderly debilitated person. This important complication has been somewhat neglected recently because of more rapid progress in the comprehension of electrolyte imbalance, pulmonary embolism and the metabolic aspects of convalescence. Nevertheless, pulmonary complications continue to plague both patients and surgeons, and this review covers recent findings in the pathogenesis and management of this important source of postoperative morbidity.

To the best of our present knowledge there are four principle factors concerned with the pathogenesis of postoperative pulmonary complications:

1. *Viscous, Mucoid Bronchial Secretion.* deTakats[17] has shown that abdominal or thoracic trauma, tugging on the cystic duct, and small pulmonary emboli all cause bronchial secretions to increase. This factor is probably present after every operation. Culver and associates,[6] in a large series of operations injected dye into the stomach before operation and carefully examined the bronchial tree postoperatively. In 8.3 per cent of these patients there was definite staining of the bronchi indicating silent aspiration with no evident vomiting. Gastric contents in the bronchial tree have been shown to set up a severe chemical reaction with profuse secretion.[15] Drinker[9] demonstrated beautifully how either inspiratory resistance or hypoxia causes a transudation into the alveoli and bronchi. Thus, a vicious cycle is established; increased secretions cause increased inspiratory resistance and hypoxia; leading to more secretions. The fact that the mucous glands of the bronchial tree produce most of the transudate means that a thick tenacious material is secreted. This inhibits one of our best mechanisms of secretion removal: the action of small cilia lining the bronchi which sweep liquid material toward the pharynx.

2. *Bronchial Obstruction Which Cannot be Relieved by Coughing.* This second factor in the pathogenesis of postoperative pulmonary difficulties is usually a direct result of the first, that is, a mechanical obstruction by viscous secretions. There may be another mechanism of obstruction, described by deTakats,[7] of reflex bronchospasm resulting from any trauma or operation or from small pulmonary emboli. Such bronchial obstruction allows air to be absorbed behind it with collapse of the alveoli, preventing adequate drainage and producing hypoventilation and pneumonia. Usually hypersecretion causes only temporary embarrassment if the patient's cough mechanism is active. The depressant effects of

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deep anesthesia or postoperative debility are factors that lead to trouble. Pain from a thoracic or abdominal incision often causes patients to repress the desire to cough. Here again a harmful cycle begins: secretions are not removed; partial asphyxia develops and a semi-comatose state with even further cough depression develops.

3. *Obstruction of Pathways of Collateral Respiration.* Van Allen and Lindskog[20] have clearly shown that small pathways, probably alveolar pores, exist between the alveoli of adjacent segments of any lobe of lung, so that obstruction of a single segmental bronchus is obstructed. Ankeney and co-authors[1] demonstrated that pulmonary hypervolemia from clamping the pulmonary veins, or from rapid intravenous infusion of whole blood rapidly decreased this collateral ventilation. The dilated pulmonary capillaries were shown to encroach on the alveolar pores, blocking their usefulness in preventing atelectasis. This mechanism undoubtedly plays a part in the *congestive atelectasis* after overtransfusion described by Jenkins, Jones, Wilson, and Moyer.[16] Stasis in the dependent portions of the lungs of patients lying motionless for long periods of time under anesthesia is another likely cause of decreased collateral respiration. Drinker and Hardenbergh[17] have demonstrated such stasis in the lungs of anesthetized animals. Because of the low pressure in the pulmonary veins, gravity has a greater effect on the pulmonary than on the systemic circulation, and greater pressure is necessary to drain blood from the parts of the lungs which are below the level of the pulmonary veins. The importance of stasis is confirmed by the fact that postoperative complications usually occur in those portions of the lower lobes caudad and posterior to the level of the inferior pulmonary vein.

4. *Infection.* Little need be said here except to emphasize that recent evidence as presented above points to the fact that infection is usually a secondary rather than a primary pulmonary complication. Pneumonitis develops in poorly ventilated areas. Purulent exudate may be even harder to remove by coughing than a viscous transudate.

PREVENTION AND TREATMENT

In preventing and treating pulmonary complications, each of these mechanisms must be considered:

1. *Viscous Mucous Secretion.* The prevention of bronchial secretion is of primary importance. Prevention of anoxia during anesthesia and maintenance of an adequate airway will reduce the transudation demonstrated by Drinker.[9] Regurgitation or silent aspiration can be reduced by emptying the stomach well with a gastric tube before surgery. Culver[6] found that regurgitation into the pharynx occurred twice as frequently and aspiration four times as often when there was no gastric tube used during operation. Routine use of preoperative atropine reduces the bronchosecretory mechanism. Early removal of nasopharyngeal and gastric tubes after operation will prevent the irritation and secretion which these adjuvants produce in the pharynx. Deep breathing exercises are important in preventing hypoventilation and anoxia.

Once excessive bronchial secretions have formed, removal by coughing or as-

piration will be assisted by decreasing the viscosity of the secretions. Baker, Roettig, and Curtis[4] have recently advocated intravenous sodium iodide in 1.0 Gm. doses twice a day. They observed the production of a thin watery secretion in 8 to 15 minutes lasting 3 to 4 minutes after injection. Examination of the iodine content of the bronchial secretion as compared with the blood iodine level at this time revealed a level 10 times higher in the bronchial secretions than in the blood. This demonstrated the selective secretion of iodine by the bronchial tree which must cause hypersecretion of fluid by either direct stimulation of bronchial glands or alteration of cellular permeability of the bronchial mucosa. Peck and Levin[9] advocate the use of an enzyme, Tryptar, to loosen viscid secretions. This enzyme is said to be effective in dissolving mucus but does not harm normal cells which contain antitrypsinoginase. Atkins[3] has found aqueous penicillin, 5000 units per cc., as an aerosol in the tracheobronchial tree to be more effective an agent than potassium iodide in liquefying secretions.

2. *Bronchial Obstruction Which Cannot be Overcome by Coughing.* The first measure of importance here is relief of pain in the chest or abdominal incision which has prevented deep breathing or coughing. Brewer, Burbank, and Samson[5] have shown that intercostal nerve block with novocaine after painful thoracic injuries has been of benefit in allowing a more effective cough. It should probably be used more in preventing pain in thoracic and upper abdominal incisions.

The bronchial spasm that occurs after injury or operation has been shown by deTakats[7] to be relaxed in only 30 per cent of experimental animals by atropine. This is not practical therapy because atropine causes secretions already present to become thick and tenacious.

If the patient cannot adequately cough up thick secretions because of debility or unconsciousness, other methods of removal must be used. The simplest is tracheobronchial aspiration with a catheter as advocated by Haight.[12] This technic is not so easy as it was at first thought to be, and it is often traumatic and painful if it must be used repeatedly. Forreger[11] has used endotracheal tubes in unconscious patients over prolonged periods, but here again, trauma to the trachea or vocal cords with later glottic edema may result. Bronchoscopic aspiration of a mucous plug is very effective if this happens to be the cause of atelectasis. Unfortunately, a single plug is too seldom the cause of trouble and thick secretions in the small bronchioles are usually found. For this reason, tracheostomy is becoming more and more commonly used in cases of severe pulmonary congestion. The advantages of tracheostomy are many; the complications are few. The nursing staff can much more easily maintain a dry unobstructed tracheobronchial tree; oxygen can be administered easily and effectively and airway resistence is decreased. In addition, the introduction of a catheter through the tracheostomy is a powerful cough stimulus. After certain operations, such as high esophageal resections and radical mandibular and neck dissections, a tracheostomy is indicated to prevent postoperative aspiration and to maintain an adequate airway if laryngeal edema or obstruction due to interstitial hemorrhage should occur. Arheleger[2] has described a new double lumen tracheostomy tube

with an inflatable balloon to prevent aspiration of gastric contents that regurgitate into the pharynx. Dickman and Baronofsky)[8] report a series of cases of severely debilitated patients who were much improved from the standpoint of pulmonary congestion after a tracheostomy was done, allowing thorough catheter aspiration of the bronchi.

3. *Maintenance of Collateral Respiration.* Maier[18] has emphasized the importance of avoiding stasis in the lungs in prevention of pulmonary complications. The aid of gravity may be obtained in draining parts of the lung below the level of the pulmonary veins by periodically turning the patient completely on the side or to a prone position. Fowler's position will prevent pulmonary vascular congestion which occurs in the supine or Trendelenberg position when blood shifts from the legs to the lungs (Hamilton and Morgan). Jenkins, Jones, Wilson, and Moyer[16] have effectively warned against overinfusion or transfusion which may cause congestive atelectasis—fatal in 7 out of their 8 patients despite oxygen, digitalis, and bronchoscopy. Holden, Cole, and Portmann[14] showed that central venous pressure rose in normal dogs during massive transfusion, but fell to normal when the transfusion was stopped. If the descending branch of the left coronary artery was ligated one month before the experiment, however, central venous pressure continued elevated after a large transfusion, and arterial pressure gradually fell until death. The lungs were intensely hyperemic. This identifies the mechanism of left ventricular failure and pulmonary edema in patients with coronary insufficiency following rapid or excessive infusion.

4. *Pulmonary Infection.* Pulmonary infection has been reduced to a position of secondary importance with the aid of antibiotics. Routine use of antibiotics after every operation will not reduce the incidence of pulmonary complications, as there are many other mechanisms of pathogenesis as pointed out above. It may, in fact, be harmful in making the tracheobronchial flora resistant to antibiotics when needed later.

SUMMARY

Postoperative pulmonary complications are produced by four principle factors:

1. Viscous bronchial secretion.
2. Bronchial obstruction that cannot be relieved by coughing.
3. Interference with collateral respiration.
4. Infection.

The pathogenesis, prevention and treatment of each of these factors is discussed.

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A CLINICAL EVALUATION OF SUCCINYLCHOLINE (WIN 7907)*

A REPORT OF ITS USE IN 100 CASES

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Anesthesiologists have long felt a need for a curare-like compound, free from side effects and combining low toxicity with an easily regulated action. Since the first use of curare by Griffith[7] in 1942, much interest has been evoked in this drug and a number of curare-like preparations have been used to advantage, although none has been free from disadvantages and side effects.

The most usual complications associated with the use of these compounds have been a fall in blood pressure, tachycardia, histamine release, increased bleeding and salivation. While these effects are uncommon, they all occur. All curare preparations have a relatively long action (15 to 40 minutes), and for this reason, when used in cases of short duration where muscular relaxation is desired, prolonged apnea and other complications may make their use difficult.

Bovet[2] (1949), in his comprehensive study of synthetic curare-like compounds, described succinylcholine iodide and noted its short term action and production of relaxation equivalent to that of curare. Stimulated by the increasing number of reports appearing in the British literature concerning the use of succinylcholine, we decided to make a clinical evaluation of a similar compound (Win 7907) supplied by Winthrop-Stearns Co.

PHARMACOLOGY

Win 7907 is a white powder readily soluble in water and has the chemical formula of succinic acid bis (B-dimethyl-aminoethyl) ester bis methochloride. Win 7907 resembled decamethonium and acetylcholine in its actions. It produces muscular paralysis by causing depolarization of motor end plates. It differs from d-tubocurarine and flaxadil. These substances produce neuromuscular blockade by lessening the depolarizing activity of acetylcholine, by raising the threshold. This explains why inhibitors of choline esterase are antagonists to these substances, Feldberg[6] 1951.

Win 7907 is destroyed by choline esterase. Neostigmine and other drugs that inhibit choline esterase prolong the action of this compound. Evans and associates[5], in 1952, found that succinylcholine is broken down in vitro and vivo by the pseudoacetylcholine esterase in the serum, although at a slower rate than acetylcholine. It is a competitive inhibitor of acetylcholine hydrolysis by both the true and pseudoacetylcholine esterase. This inhibition can be demonstrated at a concentration of 5 ug. of succinylchloride per ml. of serum. The break-down products of this hydrolysis are succinic acid and choline, both of which are natural

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metabolites. This is presumably the reason for the short duration of action (3 to 5 minutes), and decidedly low toxicity.

The HD_{50} for succinylcholine is 0.20 mg. per kg. of body weight, whereas it is 0.15 mg. per kg. of body weight for d-turbocurarine. Succinylcholine is a powerful inhibitor of neuromuscular transmission, its potency being somewhat lower than d-tubocurarine, von Dardel and Thesleff.[10]

The toxicity of repeated doses of succinylcholine has been studied in rabbits, and it was found that a daily intravenous dose of 100 ug. per kg. of body weight for five days, was enough to produce paralysis in all of them, but to cause death in none. These animals were kept under observation for eight weeks without any delayed effects being manifested.

The freedom of succinylcholine from toxicity is indicated by investigation in the dog reported by Bovet and co-workers[3] in 1951. Dogs, with ventilation maintained by a respirator, tolerated a dose of succinylcholine that is 450 times the

TABLE I

Types of cases and operative risks

Upper abdominal	22
Lower abdominal	27
Thoracic	10
E.E.N.T.	7
Dental	24
Miscellaneous	13
Class I	45
Class II	25
Class III	19
Class IV	4
Class V	5
Class VI	3

paralyzing dose, although they could not tolerate more than 15 times the paralyzing dose of d-tubocurarine.

METHODS

Win 7907 was used in 100 unselected patients requiring muscular relaxation for intratracheal intubation or operation (Table I). These patients were all premedicated with morphine and scopolamine according to age or physical condition. The age range in this group of patients was 4 to 75 years (Table II). There were 40 males and 61 females in this series of cases. Anesthesia as induced with sodium pentothal or surital in 92 cases. The average dose necessary for induction being 300 mg. Anesthesia was maintained with pentothal, nitrous oxide or cyclopropane and all patients were carried in Plane I of surgical anesthesia in order to properly evaluate the relaxing properties of Win 7907.

Intubation was accomplished after injecting intravenously 20 to 40 mg. of Win 7907. In approximately one minute after the injection, a patient was relaxed

sufficiently for intubation. The average duration of relaxation with this drug is two to three minutes. Intubation must be done rapidly, to avoid loss of muscular relaxation. In all cases relaxation was excellent. Little or no pharyngeal activity was observed; the cords were abducted, immobile, and would not respond when touched.

A continuous intravenous drip of Win 7907 is prepared by adding 400 to 500 mg. of this drug to 250 cc. of physiologic sodium chloride solution making a concentration of 0.1 to 0.2 per cent solution. This dilute solution of Win 7907 is started immediately after intubation and is allowed to flow at a fairly rapid rate (40 to 60 drops per minute) to maintain relaxation. By regulating the number of drops per minute it is easy to produce any desired degree of relaxation. This is

TABLE II

Age group distribution

0-10 Years	2
10-20	9
20-30	19
30-40	20
40-50	17
50-60	13
60-70	19
70-80	2

TABLE III

Milligrams of Win 7907 with various anesthetic agents

Cases	Anesthetic Agent	Mg. of Win 7907
39	Cyclopropane	3.8 mg.
31	Pentothal & nitrous oxide	4.2 mg.
70 cases, average amount		4. mg.
12 cases with liver damage		3.7 mg.

essentially the same technic as outlined by Bourne.[1] Table III summarizes the quantities of Win 7907 that were used to produce relaxation.

In operations of short duration, 30 minutes or less, when it is not considered necessary to prepare large quantities of dilute solution of Win 7907 this drug is injected intravenously in intermittent doses of 5 to 10 mg. every three to five minutes to produce the required relaxation.

RESULTS

Win 7907 produced adequate relaxation in all patients and was relatively free from side effects frequently seen with other relaxing drugs. Muscular relaxation could be produced easily and the degree of relaxation could be regulated from minute to minute by increasing or decreasing the flow rate of the continuous

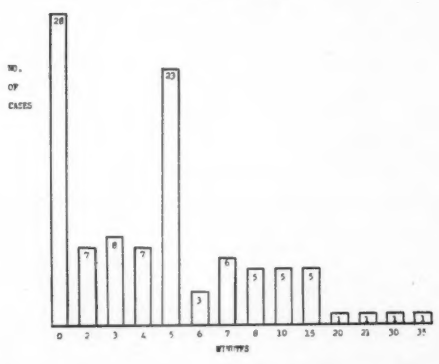
drip solution of Win 7907. The average quantity of Win 7907 necessary to maintain relaxation was 4 mg. per minute with extremes being 2 and 20 mg. per minute.

Respiratory arrest was of much shorter duration than that seen with other relaxing drugs now in use. The average duration of apnea after single injections is 5.5 minutes in 75 per cent of the patients. Twenty five per cent have mild to moderate depression of respiration. When controlled ventilation was employed, the average time for resumption of adequate tidal volume was four minutes. This time is taken from the time Win 7907 was discontinued until respiration was adequate, Table IV.

Blood pressures were taken on the ward before premedication was given; again on arrival in the operating room and immediately after injection of Win 7907. In only 2 patients did a drop in blood pressure occur that was below the level taken on the ward before premedication was given. One of these patients had a toxic

TABLE IV

Duration of Apnea



adenoma of the thyroid with a basal metabolic rate of plus 27 and was highly excited. The other patient was an extremely apprehensive white female admitted for pelvic surgery. A transient rise in blood pressure of 10 to 15 mm. systolic pressure was observed in these patients. This slight elevation of blood pressure was of short duration, 5 to 20 minutes. The blood pressure then reverted to levels obtained prior to the injection of Win 7907. The rise in blood pressure is said to be due to the *nicotinic* effect of succinylcholine and has previously been described by Bovet and associates.

No variations in pulse rate or rhythm was observed in these patients. Excessive salivation or bronchospasm was not noted. In 3 patients deliberate traction was made upon the mesentry and hollow viscera in order to observe whether vagal reflexes might be initiated. No bradycardia or change in blood pressure or respiration was noted.

It is our impression that smaller doses of pentothal were necessary when Win

7907 was used in conjunction with nitrous oxide than when pentothal and nitrous oxide was used alone. In 31 patients the average quantity of pentothal necessary for maintenance was 280 mg. with extremes of 1200 mg. in 1 patient and none in 7 patients. The average duration of operations was 100 minutes.

Recovery time from anesthesia was markedly reduced. Seventy five per cent of the patients awakened in the operating room at the conclusion of the anesthesia. These patients were fully conscious, alert and were in possession of their protective reflexes. The remaining 25 per cent of the patients awakened in 5 to 15 minutes after the anesthetic was discontinued.

Encouraged by the reduction in the quantity of pentothal required in these patients and their short recovery time, we used Win 7907 in dilute continuous drip solution in 12 patients with liver damage which was detected either by laboratory tests or clinical observation. In this group of patients 220 mg. of Win 7907 per hour was used to maintain relaxation, and pentothal and nitrous oxide or light cyclopropane were the anesthetic agents employed. In none of these patients did we encounter marked respiratory depression or prolonged duration of apnea following controlled ventilation. The recovery time, 12 minutes, in these patients did not differ significantly from the average recovery time, 8 minutes, observed in other cases without liver damage. Using minimal doses of other relaxing drugs in patients with liver disease, we have previously observed prolonged apnea following controlled respiration with long recovery time and postoperative complications.

The prompt and complete relaxation produced by Win 7907, followed by rapid recovery made this relaxant ideal for esophagoscopy and dental extraction. By using this agent we were able to produce adequate relaxation, and avoid deep anesthesia and prolonged recovery time. These procedures can be done safely on out-patients, saving bed space and expense.

CASE REPORT

The patient was a 69 year old white man whose history dated back to Sept. 1, 1952 at which time a diagnosis of lymphosarcoma was made. This patient had been treated with deep roentgen ray therapy and was readmitted to this hospital on Dec. 3, 1952 with a history of severe hematemesis and melena of three days duration prior to admission.

The initial blood count revealed red blood cells 1,220,000 per cu.mm., white blood cells 16,600 per cu.mm. and a hemoglobin of 3.2 Gm. per 100 cc. (20.7 per cent). The blood urea nitrogen was 64.5 mg. per cent.

He was given 5,000 cc. of blood and an exploratory laparotomy was done on Dec. 9, 1952. At this time the red blood count was 2,600,000 per cu.mm. with hemoglobin 7.7 Gm. per 100 cc. (50 per cent).

Atropine gr. $\frac{1}{100}$ was given one hour prior to operation and the blood pressure taken in the operating room was 130/80. Anesthesia was induced with cyclopropane and as soon as the patient was unconscious, a continuous drip of Win 7907 was started at the rate of 60 drops per minute. Within three minutes after beginning the Win 7907 infusion, intubation was done with ease. Five hundred cc. of blood was started immediately. The blood pressure taken immediately after intubation was 160/40 and the pulse rate was 102 per minute.

After slowing the Win drip to 30 drops per minute, respiration was of adequate tidal volume in five minutes with a respiratory rate of 24 per minute. Blood pressure was maintained at 140 to 160 systolic and 90 to 100 diastolic during operation with a pulse rate of

100 per minute and a respiratory rate of 20 to 24 per minute. Anesthesia was maintained with light cyclopropane and Win 7907 was given at the rate of 16 to 60 drops per minute to provide relaxation. A total of 400 mgs. of Win 7907 was given during operation.

At operation, multiple ulcerated lesions were found in the mucosal surface of the stomach and it was decided that these ulcerations were causing the bleeding. A gastric resection was done, resecting the distal two thirds of the stomach. The operation was of two hours duration, and 15 minutes after conclusion of the operative procedure, the patient was wide awake and was talking. A liter of blood was given during the operation.

The postoperative course was uneventful. The blood pressure was maintained at 140/90 and the patient was afebrile on the second postoperative day. He was discharged on the eleventh postoperative day, with a red blood count of 4,360,000 per cu.mm. and a hemoglobin of 12.4 per cent.

We have chosen this case as an illustrative example to show what can be accomplished with the use of succinylcholine in extremely poor-risk patients. We believe that Win 7907, in combination with cyclopropane, is the drug of choice in this type of patient, since with this combination we can maintain a light plane of anesthesia, and produce minute to minute control of relaxation, and avoid respiratory and cardiovascular depression.

DISCUSSION

In our hands Win 7907 is far superior to other relaxing drugs now in general use. It provides moment to moment control of relaxation and, by adjusting the drip rate, we are able to alter muscular relaxation whenever necessary.

Respiratory depression was of shorter duration than with other drugs now in general use. In only 4 patients was prolonged apnea encountered. Prolonged apnea in these patients was of 15, 21, 30 and 35 minutes duration. Three of these patients had thoracic operations and controlled ventilation was used. This technic undoubtedly was responsible for the production of apnea, due to depletion of blood carbon dioxide.

Lehmann[8] has reported respiratory arrest for six and one-half hours after injection of 50 mg. of succinylcholine. His patient received neostigamine which probably prolonged the apnea since it is a powerful inhibitor of choline esterase. Evans[5] and co-workers have measured acetylcholine esterase blood levels and found that in their 6 cases with prolonged apnea, low levels were obtained. They noted patients having carcinoma, severe anemia, liver disease or poisoning with anticholine esterase compounds also had low esterase blood levels. Love[9] has also reported a case of prolonged apnea of 50 minutes with convulsions due to hypocalcemia which responded to intravenous calcium. He concluded that patients with a tendency to muscular spasms due to such conditions as tetanus and hypocalcemia should not be given succinylcholine. Ellis[4] and associates have observed that succinylcholine is less likely to cause central respiratory depression than d-tubocurarine, syncurine and flaxadil.

It is our opinion that, while there may be rare occurrences of prolonged apnea, we should not condemn this drug, but such occurrences should serve to make anesthesiologists aware of sensitivity and prevent overdoses and administration of choline esterase inhibitor drugs.

The absence of side effects with Win 7907 was striking. The occasional muscular twitching that occurs after rapid administration is due to the initial stimulation of depolarizing drugs on skeletal musculature and is of no consequence.

In our hands Win 7907 has been a very useful muscle relaxant. It is our drug of choice for endotracheal intubation and for the production of relaxation for operations of short duration. We have not observed adverse side effects. Debilitated poor-risk patients have well tolerated this drug, when combined with minimal quantities of anesthetic agents.

SUMMARY

Win 7907 was used in 100 unselected patients requiring relaxation for intratracheal intubation or operation.

Adequate muscular relaxation was produced by Win 7907 and the degree of relaxation could be regulated from minute to minute.

Respiratory arrest was of shorter duration than that seen with other relaxing drugs now in general use.

It is our impression that smaller doses of pentothal were necessary when Win 7907 was used in conjunction with pentothal and nitrous oxide.

Recovery time from anesthesia was markedly reduced.

This drug was well tolerated by patients with liver damage.

The absence of side effects with the use of Win 7907 was striking.

Combined with minimal quantities of anesthetic agents Win 7907 was well tolerated in debilitated poor-risk patients.

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SURGICAL TREATMENT OF STENOTIC VALVULAR DISEASE OF THE HEART*

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Access to the interior of the heart has been the object of surgical studies for many years. The development of pumps and oxygenators for extracorporeal circulation,[22, 23, 31] cooling to low temperatures for the purpose of decreasing heart rate and oxygen requirement,[6, 8, 19, 34] and the use of an auricular well for entrance into the heart[28] are all promising methods for making open surgery of the heart possible. However, concomitant with these approaches to the problem, closed methods of correcting certain defects have been developed. The two which have proved most useful and practical are mitral and pulmonary valvulotomy. The present status of these procedures will be considered in this report.

Fifty years ago Samways,[43] Brunton,[14] and their contemporaries[21] suggested that mitral stenosis be treated by surgical methods. Later Allen and Graham[1] devised a cardioscope through which they operated on the mitral valve via the auricular appendage in 22 dogs with but one fatality at the time of operation. However, the only trial in a patient was a failure.[21] Subsequently Harken[29] and Brock[9] also abandoned the use of a cardioscope as impractical. Cutler, Levine, and Beck[21] were the first to give valvulotomy for mitral stenosis an extended trial and accumulated 7 cases. The first case operated upon survived four and one-half years, but the remainder of their patients died early in the postoperative period. The cardiovalvulotome devised by Beck and Cutler was used by Pribram[42] in 1 case, but the patient died on the sixth day.

In 1925 Souttar[47] introduced his left index finger into the left atrium through an opening in the appendage of a patient with mitral stenosis and regurgitation. Because of regurgitation and a moderate degree of stenosis he did not use the thin hernia bistoury he had available to pass along his finger and cut the valve but dilated it with his finger. The illustrations in his paper, showing the use of the auricular appendage as the point of entrance, a soft-bladed clamp placed at the base of the appendage for hemostasis, and the introduction of a knife guided by the finger, are remarkably similar to present day methods of attack on the mitral valve. His words are also prophetic:

"It appears to me that the method of digital exploration through the auricular appendage cannot be surpassed for simplicity and directness. . . . Owing to the simplicity of the structures, and, oddly enough, to their constant and regular movement, the information given by the finger is exceedingly clear, and personally I felt an appreciation of the mechanical reality of stenosis and regurgitation which I never before possessed. To hear a murmur is a very different matter from

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feeling the blood itself pouring back over one's finger. I could not help being impressed by the mechanical nature of these lesions and by the practicability of their surgical relief." It is unfortunate that his case was not more suitable.

Harken,[29] Smithy,[46] Sweet,[48] and others continued to seek a surgical solution to the problem of mitral stenosis. Reports of greatest success did not appear, however, until after Bailey[2] opened the lateral commissure of a 24 year old woman with mitral stenosis by means of a finger knife on June 10, 1948. Utilizing this concept of commissurotomy, Bailey, Glover, and co-workers and many others have also obtained very gratifying results since that time. Additional experience has shown that the use of a knife is not necessary in every case but the commissures can often be fractured with the finger.[30] The treatment of insufficiency of the mitral valve with flap valves[3, 36] awaits further evaluation.

In 1913 Doyen[24] attempted to treat pulmonic stenosis by incision with a tenotomy knife, entering the heart through the right ventricle. The patient survived only a few hours and at autopsy was found to have subvalvular stenosis, interventricular septal defect, and incomplete development of the lung. Sellors[44] opened a stenotic pulmonary valve successfully with a long tenotomy knife in a boy 20 years of age with pulmonary tuberculosis and a clinical picture of tetralogy of Fallot on Dec. 4, 1947. Shortly thereafter, Brock[9] also did a pulmonary valvulotomy with survival in an 18 year old girl with tetralogy of Fallot and valvular stenosis. Subsequently he experienced similar success in operations for pulmonary valvular stenosis not only in England[10] but in this country[7] as well. Infundibular stenosis presents a more difficult technical problem, but Brock[10, 11, 13] and Glover and associates[25, 26] believe that resection of the occluding tissue through the right ventricle is superior to the construction of extracardiac shunts in most cases. Blalock and coworkers[32] and Potts[40] are more conservative in their viewpoint. Although a number of methods have been used to correct aortic stenosis from the time of Tuffier's often-quoted attempt to the experience of Bailey, Ramirez, and Larzelere[5] who dilated the valve through the ventricle with a special instrument, no universally accepted procedure has emerged.

INDICATIONS FOR MITRAL COMMISSUROTOMY

At this time only patients with functional capacity rated in classes II, III, and IV are accepted for surgery, and operation is not advised in those without symptoms. The anatomical situation most desirable is the presence of pure stenosis with no associated valvular lesion and cusps which are mobile at the base. Calcification of the valve is not a contraindication but may complicate the procedure, and the results may not be as good when the disease is that far advanced. If present, regurgitation should be minimal, but operation may be beneficial in the presence of regurgitation in selected cases. The presence of a systolic murmur is not always a reliable indication of mitral regurgitation, and occasionally it is necessary to wait until the finger is in the left atrium before making a decision about the advisability of commissurotomy.

A history of emboli makes the prognosis poorer but does not contraindicate

operation. On the contrary, operation is more urgent in these patients. Likewise auricular fibrillation, hemoptysis, and pregnancy do not preclude operation. Operation is seldom in order in the presence of left ventricular enlargement, but this enlargement may sometimes be difficult to detect. The electrocardiogram can be helpful in this regard, especially when a left axis shift is present in the absence of extra-cardiac modification of the tracings. The optimum age is usually 20 to 40 years, but the patient's condition is used as a basis for decision rather than any arbitrary age limit. Operation is not done in the presence of acute rheumatic disease and subacute bacterial endocarditis.

OPERATIVE PROCEDURE IN MITRAL STENOSIS

Unless the patient is in failure, digitalis is not administered before operation, and quinidine is not given routinely, but penicillin administration is always started the day before operation. Cyclopropane and ether are the anesthetics used most frequently by Dr. Adriani and his staff. The chest is opened through a posterolateral incision (fig. 1), with removal of the fifth rib. After the thoracic cavity is entered, adequate exposure is obtained by using a Burford retractor. Incision is made in the pericardium over the auricular appendage. This incision is usually placed anterior to the phrenic nerve, although on some occasions it seems more advantageous to place it posterior to the nerve (fig. 1). The incision is not extended the full length of the ventricle in order to avoid as little change in position of the heart as possible. Great care is taken to ligate all bleeding points encountered. A suture placed in the pericardium is useful for retraction as it is incised, and traction sutures through the cut edges of the pericardium also provide more satisfactory exposure.

A purse-string suture of no. 00 Deknatel silk is placed in the base of the auricular appendage, bringing the ends out through a glass tube or more elaborate Rumel-Belmont tourniquet so the second assistant can tighten it at the proper time and yet have his hand well out of the operative field. The appendage is manipulated as little as possible until a clamp is placed across its base because of the danger of dislodging thrombi which may be present within the appendage.

After a noncrushing clamp is applied to the base of the appendage (fig. 2) between the ventricle and purse-string suture, taking care not to damage adjacent coronary vessels, an incision is made in the lateral wall of the auricular appendage of sufficient length to admit the index finger with a snug fit. If thrombi are encountered, the clamp is opened sufficiently to allow loose fragments to be washed out through the opening. Trabeculations across the appendage are cut, and the index finger of the right hand is then inserted into the left auricle. This maneuver is accomplished by inserting the finger as the first assistant exerts counter traction with a Babcock clamp on the auricular appendage; the second assistant draws the purse-string suture up snugly against the finger; and the operator releases the soft-bladed clamp. Emboli from the appendage are a real danger.[20] Brief occlusion of the left common carotid and innominate arteries when there is greatest likelihood that emboli may be dislodged has been advocated by Bailey and associates[4] to prevent this catastrophe.

The left atrium is explored with the finger to determine the condition of the mitral valve and the amount of regurgitation, if present. There is no need for undue haste, except for the period when the mitral orifice is occluded. The usual organ encountered tolerates this probing surprisingly well. The mitral orifice

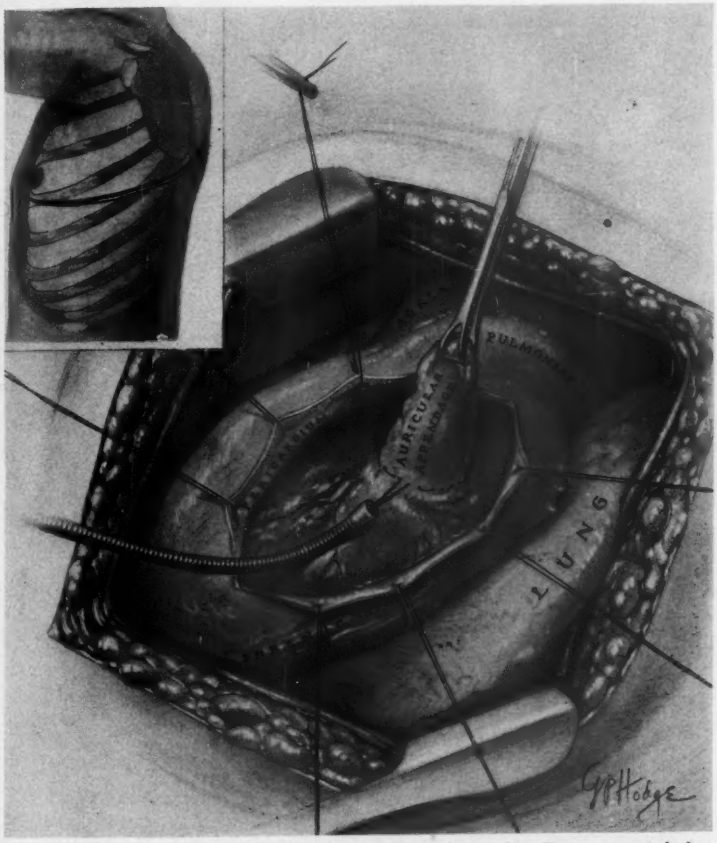

FIG. 1. Mitral Commissurotomy. This figure shows the pericardium open and the edges retracted by means of stay sutures. A Babcock clamp steadies the auricular appendage while a purse-string suture of no. 00 silk is placed at the base of the appendage. The purse-string suture is shown passing through the tip of a Rumel-Belmont tourniquet. Care is taken at this stage of the procedure not to dislodge any thrombi within the appendage. Insert shows position of posterolateral incision.

can only be obstructed for a few moments at a time, however. Fracture of commissures with the finger[30] is usually begun by pressing laterally and posteriorly, thus attacking the lateral commissure first. There is greater danger on the medial side because of the proximity of the aortic valves which may be injured. In the most favorable cases the stenosis is due to a fibrous ring about the orifice with

fusion of the commissures, but with the base of both leaflets still relatively pliable. As pressure is exerted this ring is broken and the commissure is reopened out to the myocardium. Although the additional circumference of the mitral orifice

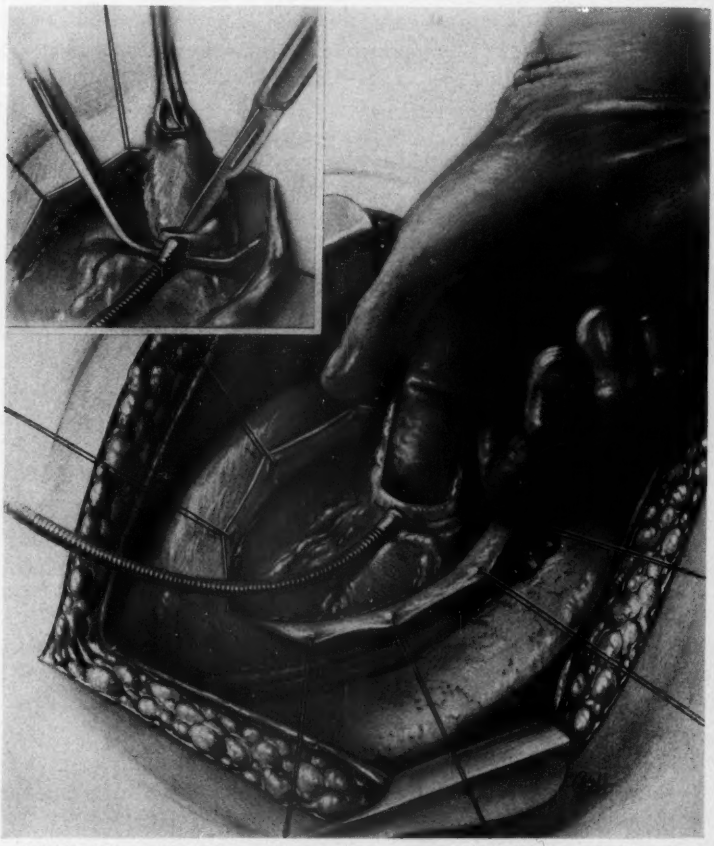

FIG. 2. Mitral Commissurotomy. Insert shows noncrushing clamp occluding the base of the appendage while an incision is being made in the lateral wall. The main figure shows the index finger of the right hand inserted in the opening through the auricular appendage into the left auricle. The purse-string suture is drawn up snugly against the finger to prevent hemorrhage. The finger has been inserted by tightening the purse-string suture and releasing the clamp as the finger was introduced into the opening and counter traction was made on the auricular appendage which has not been completely severed. Pressure toward the commissures and posteriorly may effect a commissurotomy by the finger-fracture method.

which is gained by the fracture may not be large, the opening of this fibrous and calcific ring increases the function of the valve to a relatively greater extent. The commissurotomy need not be done in a single attempt, but the finger may be withdrawn; the presence of regurgitation tested; and the commissurotomy carried out in stages over a period of several minutes. After a lateral or medial com-

missurotomy has been completed, a decision is then made as to whether it should also be attempted on the opposite side.

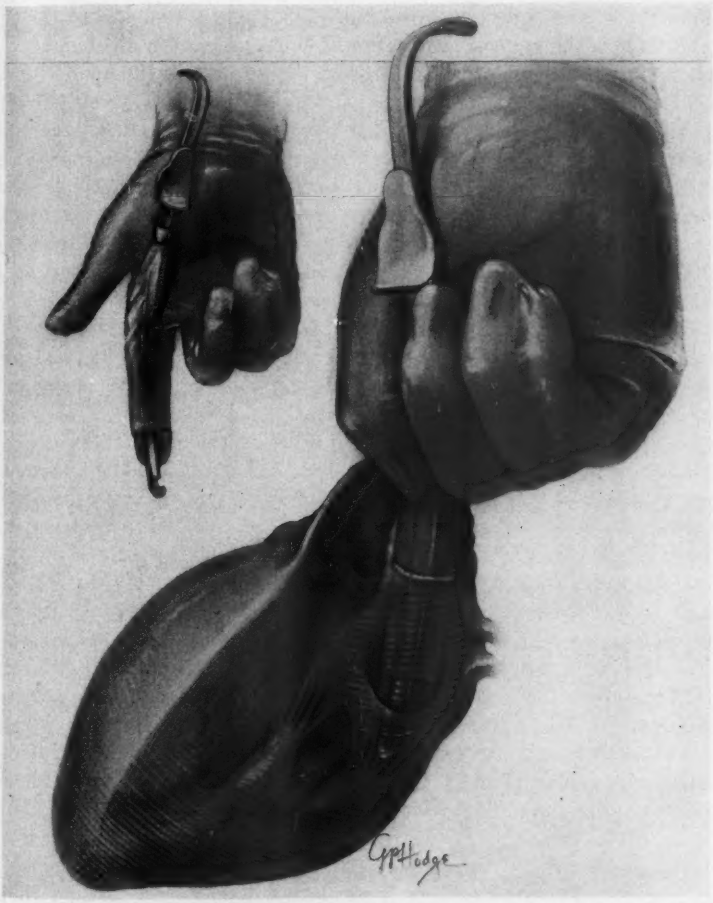

FIG. 3. Mitral Commissurotomy. This figure illustrates valvulotomy for mitral stenosis by means of a finger knife inserted between two gloves. The index finger of the outer glove is used as a point of entrance for the knife and is tied with a suture around the knife. The knife extends downward through the third finger of the outer glove which has been placed on the index finger of the hand. Two fingers of the hand are inserted in the fifth finger of the glove. The transparency of the heart indicates the position of the finger knife within the left auricle and ventricle. Closing the blades of the knife along the dotted line results in enlargement of the mitral orifice by reconstituting the lateral commissure.

If the attempt to do a finger fracture is unsuccessful, the finger is withdrawn from the left auricle, placing the soft-bladed clamp in its previous position as the finger is withdrawn and the purse-string suture is released. A second glove is drawn over the right hand of the operator as shown in figure 3. We find that the amount of bleeding is reduced if the end of the index finger of the glove is opened

and used for introducing the valvulotome between the two gloves. The outer glove is placed on the hand so that the fourth and fifth finger of the hand are inserted in the fifth finger of the glove to provide the free tube for introducing the Bailey knife. The outer glove over the index finger is removed at the tip to allow the blade of the knife to protrude. The choice of right or left-handed blade depends upon the commissure to be cut and the position of the hand necessary to effect the division of the commissure. The index finger is then reinserted into the left auricle in the manner described above. The palpating index finger guides the blade of the knife until it is properly engaged, and the incision, shown as a dotted line in figure 3, is made by closing the engaging blade with the free hand. After the incision is made, the knife can be drawn up slightly above the tip of the finger which is then used to gauge the adequacy of the opening and the amount of the regurgitation. When valvulotomy is complete the finger is withdrawn, and at the same time the noncrushing clamp is closed around the base of the appendage so that there will be minimal blood loss. It is very important to have at least two noncrushing clamps available so that it will not be necessary to remove the first clamp while the second is being applied should bleeding occur in the auricular wall on the ventricular side of the clamp.

The appendage is then excised (fig. 4A) since experimental and clinical evidence indicate that extirpation probably is without effect and the auricular appendage is the most frequent site of thrombi in rheumatic heart disease. The tissue removed is used for metabolic and microscopic studies.[16, 17] The stump of the appendage is closed with a layer of interrupted, simple sutures of no. 000 silk (fig. 4B) and a final continuous suture of the same material (fig. 4C). From studies on dogs,[15] we concluded that a linear closure of this type is superior to closure with a purse-string suture alone. The pericardial cavity is then closed very loosely with interrupted, no. 000 sutures of silk in order to allow drainage (fig. 4D), and tissue for biopsy is removed from the lung for study of pulmonary vascular changes. A fenestrated catheter is anchored to the chest wall by means of a pleural tunnel[18] and is drawn through a stab wound in the eighth interspace in the posterior axillary line. This is connected to constant suction at the end of the operative procedure. The chest is closed in layers with interrupted sutures of silk.

We do not use topical drugs during the operation unless arrhythmia is pronounced or troublesome. Procaine (2 per cent), 10 per cent calcium chloride, epinephrine, and a defibrillator are always available in the operating room. The anesthetist uses every means at his command to avoid anoxia at any time during the operation, which we believe is most important in avoiding cardiac arrest.

CASE REPORT

A 36 year-old, white housewife was admitted to the Charity Hospital for the first time on May 16, 1951. She was unaware of cardiac disease until 6 years previous to admission at which time a male infant was delivered following an uneventful pregnancy, and she was told that she had valvular cardiac disease. Two years later she began having attacks of pneumonia several times each year necessitating hospitalization on several occasions. Chills, fever, and malaise characterized these attacks. Later she noticed dyspnea and in-

creasing fatigability. Following her last attack of pneumonia, one month previous to admission, she continued to have a low-grade fever and productive cough. In early childhood the patient had chills and fever unaccompanied by jaundice, arthritis, or subcutaneous nodules. A diagnosis of malaria was made at the time. The attacks did not recur following tonsillectomy at the age of 11 years.

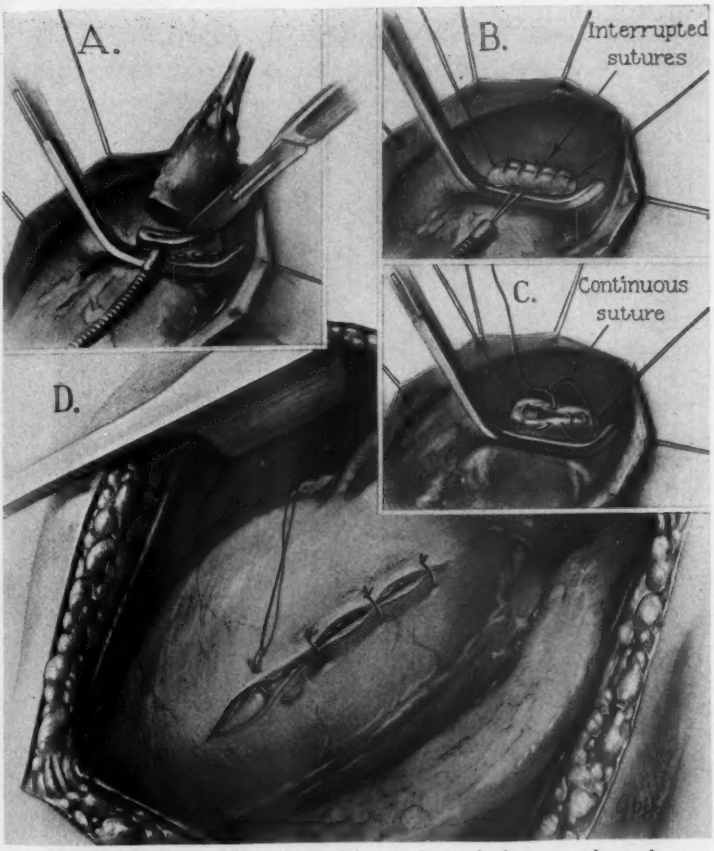

FIG. 4. Mitral Commissurotomy. A. Excision of the auricular appendage after commissurotomy has been completed.
B. First row of interrupted sutures closing the stump of the auricular appendage.
C. Final continuous layer of sutures completing the closure of the left auricle.
D. Closure of the pericardium with very few interrupted sutures to allow free drainage of fluid from the pericardial cavity.

Physical Examination: The patient's general appearance was not unusual. Her temperature was 98.6 F.; pulse, 110; respiration, 20; and her blood pressure was 144/90. The chief abnormal physical findings were confined to the heart. The point of maximum impulse was in the left fifth interspace. There was an associated diastolic thrill and murmur in the mitral area which was heard over the precordium, in the axilla, and was also audible over the posterior lung fields. Some observers heard a systolic murmur at the base. The pul-

monary second sound was louder than the aortic second sound, and a definite gallop rhythm was present. The liver was not palpable, and there was no cyanosis, clubbing of fingers, or dependent edema.

Laborating Findings: Blood Count: hemoglobin: 13.4 gm., red blood cells: 4,500,000, hematocrit: 40, white blood cells: 5,000.

Urine: Yellow, neutral, specific gravity: 1.009, albumin: negative, sugar: negative, microscopic: rare epithelial cells.

Three blood cultures, two sputum cultures, two stool cultures, and one urine culture were negative.

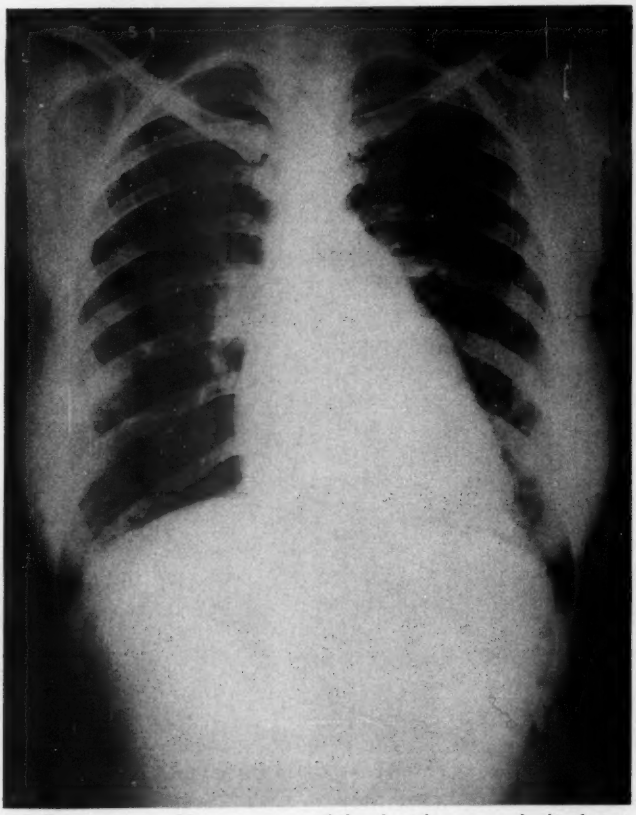

FIG. 5. Roentgenographic appearance of the chest in a case of mitral stenosis.

Electrocardiogram: Right axis deviation, P-1, 2, slightly wide, suggestive of rheumatic mitral stenosis.

Roentgenograms and Fluoroscopy: Increased fulness of pulmonary region with small aortic arch and enlargement of the right ventricle. The left ventricle was not enlarged (fig. 5).

Cardiac catheterization was done on May 16, 1951 in the Heart Station at Charity Hospital and the results may be seen in table 1 and figures 6 and 7. The patient had elevation of pulmonary capillary, pulmonary artery, and right ventricular pressures. Following a

transfusion of 500 cc. of blood in 4.5 minutes there was increased pressure in the pulmonary artery and the right ventricle and no change in the pulmonary capillary pressure. The patient was advised to have a mitral commissurotomy.

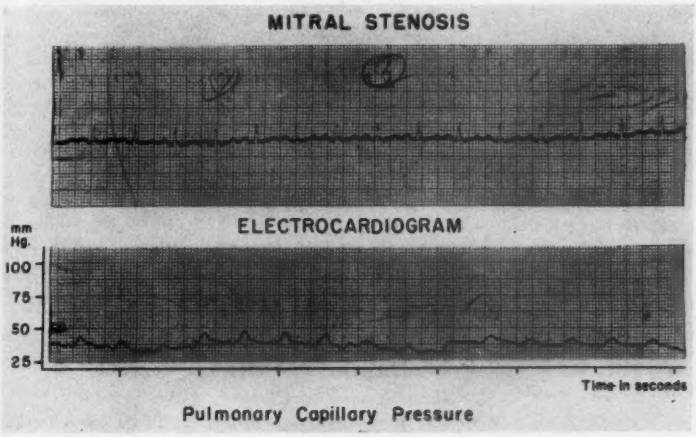

FIG. 6. Tracings of pressure in the pulmonary capillary region in a case of mitral stenosis before operation.

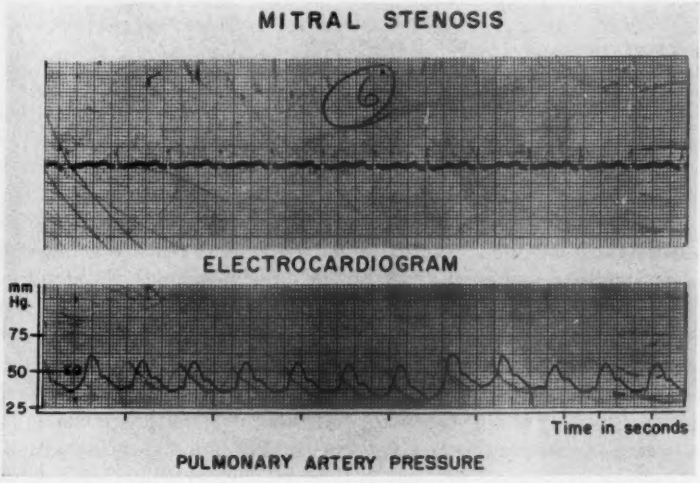

FIG. 7. Tracings of pressure in the pulmonary artery in a case of mitral stenosis before operation.

Operation: Operation was done on Sept. 19, 1951, using the technic described above. Attempts to fracture the commissures with the finger were unsuccessful, and the lateral commissure was cut with a finger knife. No regurgitant jet of blood was palpable afterward, and it was judged that an adequate opening had been made.

Postoperative Course: The patient's course was smooth until the evening of the second

postoperative day when the pulse rose from 82 to 175. She was digitalized slowly with lanatoside C, and her pulse decreased to 119 over a period of five hours. An electrocardiogram showed auricular fibrillation and right bundle branch block. On the third postoperative day the patient's rhythm reverted to a regular mechanism following administration of quinidine. After two days quinidine was discontinued, but digitalis was continued throughout the remainder of the patient's hospital stay and for two weeks thereafter. The pulse promptly returned to normal rate after conversion to sinus rhythm, and no further episodes of arrhythmia occurred. She received penicillin, 300,000 units per day, in the postoperative period. Her sutures were removed on the sixth postoperative day, and she was discharged 13 days after her operation.

After operation the diastolic murmur was audible but diminished in intensity. She improved rapidly both subjectively and objectively, and had no recurrence of dyspnea and pneumonitis. She returned to the hospital and was catheterized again on July 10, 1952. At that time the mean pulmonary capillary pressure was 27.5, and the mean pressure in the pulmonary artery was 34 (table 1). It is evident that these pressures and the arteriolar resistance have fallen when they are compared to preoperative measurements. Although not normal, these findings do represent improvement. Eighteen months after operation the patient continues to be asymptomatic.

TABLE 1

Cardiac catheterization

Mitral stenosis

	Mean Pressure (mm. Hg)		Pulmonary Arteriolar Resistance (dynes sec. cm.$^{-5}$)	Cardiac Output (liters/min.)
	Pulmonary Artery	Pulmonary Capillary		
May 16, 1951 (before transfusion)	44	36.5	143	4.2
May 16, 1951 (after rapid transfusion)	50	36.0	271	4.4
July 10, 1952	34	27.5	108	4.9

PULMONARY VALVULAR STENOSIS

When valvular stenosis occurs with an intact interventricular septum, the degree of cyanosis usually depends upon the amount of shunt through the foramen ovale from right-to-left. Blalock and Kieffer[7] report that such a shunt is present in 70 per cent of cases. Cyanosis is rare before the first month and may appear at the time of closure of the ductus arteriosus. Dyspnea and exercise intolerance frequently may be present and may be out of proportion to the degree of cyanosis. Clubbing of fingers and toes often occurs, and the liver may be large and pulsating. As failure progresses, ascites and edema may be a terminal feature. Albuminuria is found frequently. Polycythemia and prolonged circulation time depend upon the extent of right-to-left shunt.

Right ventricular hypertrophy is present, and a precordial heave and chest deformity may occur. A murmur may be heard in the pulmonary area, a thrill is often palpable, and the pulmonary second sound is usually pure. Roentgenograms and fluoroscopy show right ventricular and auricular enlargement and prominent pulmonary artery, whereas pulmonary vascular markings are dimin-

ished. Electrocardiograms show right ventricular hypertrophy. Angiograms may reveal retarded flow through the heart, enlarged right ventricle, and poststenotic dilatation of the pulmonary artery. High pressure, which may be higher or lower than systemic pressure, is recorded in the right ventricle, and low or normal pressure in the pulmonary artery. The pulmonary flow is diminished. Arterial oxygen unsaturation depends upon the shunt from right to left auricle. Although infundibular stenosis is common, valvular stenosis occurs in tetralogy of Fallot.[10, 11, 13, 25, 26, 32] When present in that condition, surgical treatment is the same as for pulmonic stenosis with intact interventricular septum.

PULMONARY VALVULOTOMY

The thoracic cavity is entered through a lateral or an antero-lateral incision (fig. 8). In adults a rib may be removed, but in children it has been customary to carry the incision through the fourth intercostal space. The pericardium is incised anterior to the phrenic nerve, and a flap is made which is hinged anteriorly, exposing the main pulmonary artery, and the outflow tract of the right ventricle. Traction on the anterior flap of pericardium tends to deliver the site of incision into the operative field. The pulmonary artery is then carefully inspected and palpated. Frequently it is possible to palpate the stenosed valve and to feel the stream of blood within the vessel as it is ejected with each systole. Poststenotic dilatation of the pulmonary artery is also characteristic of pulmonary valvular stenosis and may be quite striking.

A site for incision is selected proximal to the valve on the anterior surface of the right ventricle in the infundibular region. Stay sutures of no. 000 silk are placed on each side of the proposed incision as shown in figure 8. Using a scalpel with a small blade, an incision is then made in the myocardium of the right ventricle almost to the depth of the endocardium. The Potts valvulotome[41] with retractile blades is then introduced into the opening, first checking the width to which the blade should be opened on the calibrated scale located on the handle of the instrument. Bleeding around the valvulotome or from the opening in the right ventricle may be controlled by crossing the stay sutures and exerting traction, as shown in the figure. This traction should be very gentle, and occasionally pressure with the finger over the incision is a wiser maneuver. As soon as the valvulotome is within the chamber it is passed backward in the direction of the right ventricle in order to rule out the presence of infundibular stenosis. A curved probe is sometimes a more satisfactory and safer instrument to ascertain the site of stenosis (fig. 8).

The valvulotome is inserted into the right ventricle in the position shown in figure 9A. The blades are opened; the calibrated scale on the handle of the instrument is consulted; and the blades are then thrust through into the pulmonary artery, cutting the stenosed valve and converting it into a bicuspid valve (fig. 9B). The blades are then closed and the valvulotome withdrawn. At that point a Potts dilator is passed through the stab wound into the right ventricle until it assumes a position shown in figure 9C. It is opened gradually and passed back and forth through the enlarged orifice until the desired dilatation has been accomplished

(fig. 9D), after which it is collapsed and removed. The design of the instrument permits circulation of blood through the pulmonary vessel while the valve orifice is being dilated. Two or three interrupted sutures of no. 000 silk and a running suture of no. 00000 silk (fig. 9E) are used to close the opening in the ventricular wall.

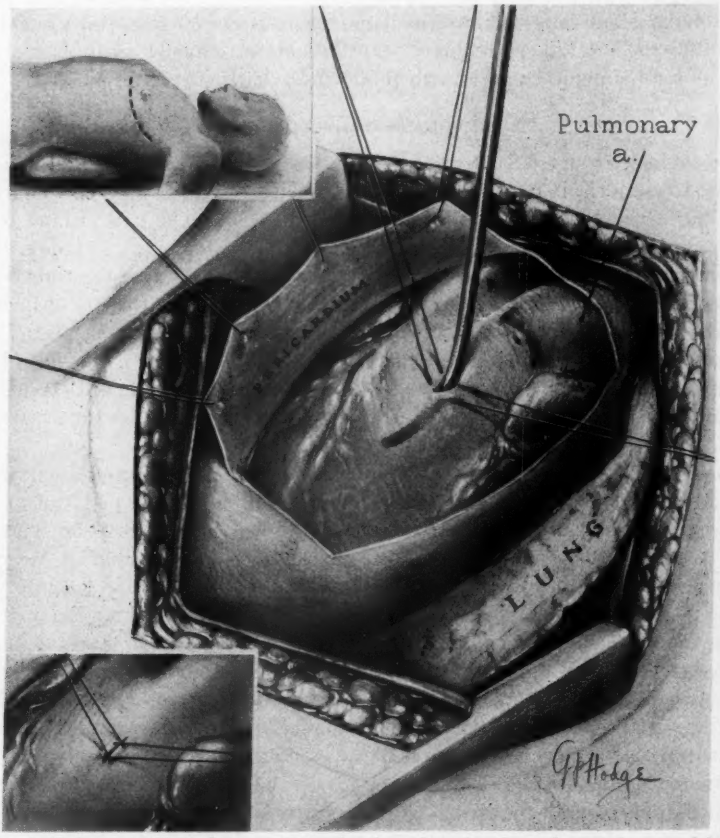

FIG. 8. Pulmonary Valvulotomy. Introduction of probe through a stab wound in the right ventricle in order to determine the site of stenosis. The upper insert shows the location of the incision. The lower insert illustrates the method of crossing stay sutures on either side of stab wound in right ventricle for hemostasis.

The entire operative procedure requires a very short time, and there is usually very little loss of blood. The pericardium is closed loosely, and the chest is closed in layers with interrupted sutures of silk and drained by means of a fenestrated catheter placed in the chest through a stab wound in the eighth interspace. At the time of operation this tube is clamped and later attached to closed suction when the patient arrives in the recovery room. It is usually removed within 24 to 48 hours. Although results of this operation are frequently dramatic, the pa-

tient's condition may become precarious before the valve is cut. If heart action becomes poor, the operator should hasten to accomplish the valvulotomy rather than wait for the myocardium to recover. When valvular stenosis is suspected in tetralogy of Fallot, it is worthwhile to open the pericardium and inspect the

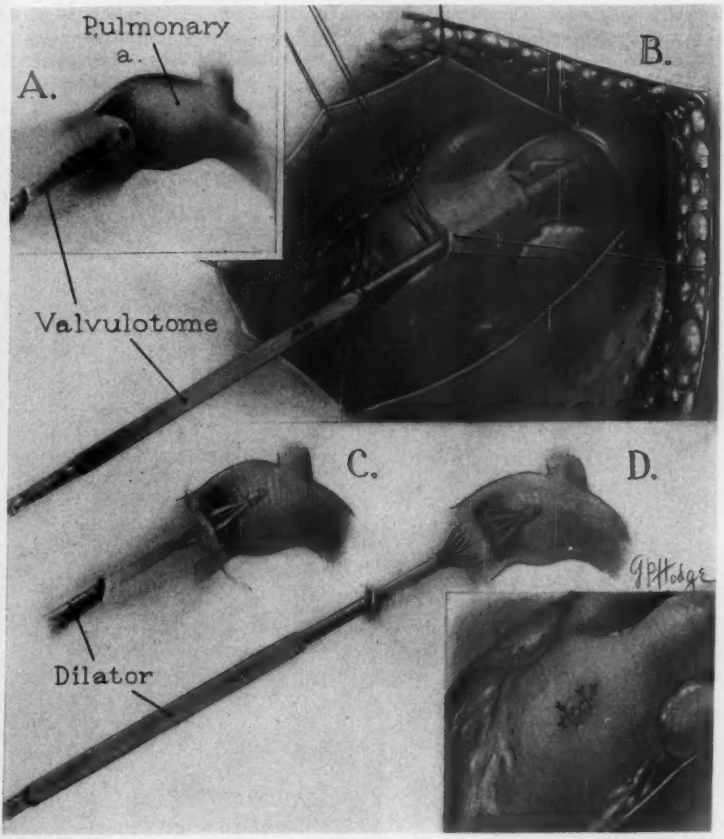

FIG. 9. Pulmonary Valvulotomy. A. Valvulotome with blade closed and tip engaged in opening of pulmonary valve.
B. Valvulotome with blades open immediately after pulmonary valve has been incised.
C. and D. Dilator gradually being opened as it is passed back and forth through the enlarged pulmonary orifice.
E. Closure of wound in heart with interrupted sutures of no. 000 silk and a continuous suture of no. 00000 silk.

pulmonary artery and infundibulum, cutting the valve if it proves to be the site of obstruction.

CASE REPORT

This patient was first seen on Feb. 10, 1949. At that time she was 24 years of age. She had been cyanotic since birth but the cyanosis was not pronounced until after an attack

of scarlet fever at the age of 14 months. Notwithstanding this cyanosis, which became deeper on exercise, she had no illnesses other than measles, mumps, varicella, pertussis, and scarlet fever during her childhood and up to the time of admission to the hospital had never found it necessary to take digitalis. Severe epistaxes began four years before admission and were the reason for seeking medical advice which led to her admission to the hospital.

Physical Examination: The patient presented a striking appearance because of intense cyanosis with brown, mottled discoloration of the legs and injection of the sclerae. The cyanosis became deeper on exercise. Her exercise tolerance, however, was impaired only moderately. Her temperature was 98.6 F.; pulse, 68; respiration, 20; and blood pressure in the arms was 130/110 and in the legs was 150/100. The maximum apical heart beat was located in the sixth interspace on the left 2 cm. outside the midclavicular line. A loud,

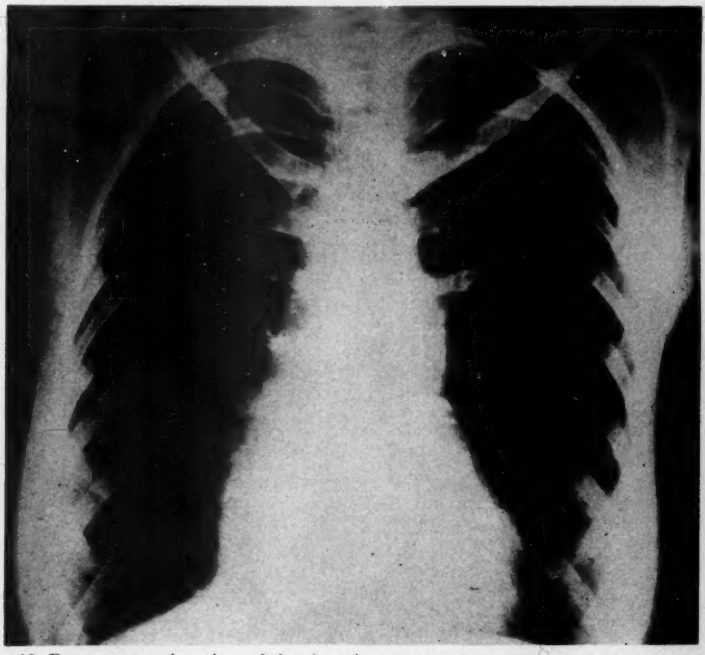

FIG. 10. Postero-anterior view of the chest in a case of pulmonary valvular stenosis with intact interventricular septum.

harsh, blowing systolic murmur was easily audible over the entire precordium, but was loudest in the second and third left intercostal spaces at the sternal border. This murmur was transmitted to the left axilla and to the neck, and could be heard posteriorly as well. The pulmonary second sound was louder than the aortic, but was diminished in intensity. The liver was not palpable. Pronounced clubbing of the fingers was present, and the nail beds were intensely cyanotic.

Laboratory Findings: Blood Count: hemoglobin: 22 gm., r.b.c.: 7,550,000, w.b.c.: 6,950.

Urine: Dark yellow, acid, albumin: 2+, sugar: 0. Microscopic: 4–5 white blood cells, occasional red blood cells and granular casts per high power field.

Roentgenograms of the chest: Slight enlargement of the cardiac shadow, prominence of the pulmonary region, suggestive enlargement of the right ventricle and diminished vascular markings (fig. 10).

Fluoroscopy: Considerable right ventricular enlargement, very slight left ventricular enlargement, enlargement of the right atrium, dilatation of the pulmonary conus, and diminished vascular markings and pulsation. It was thought at the time of fluoroscopy that pulmonary stenosis with poststenotic dilatation of the pulmonary artery could account

TABLE 2

Cardiac catheterization

Pulmonary valvular stenosis

Location	Gas	Pressure	O_2 Content
Brachial artery	Air	106/84	20.5
Pulmonary capillary	Air	15	
Pulmonary artery	Air	15	11.0
Right ventricle	Air	125/22.5	10.4
Pulmonary vein	Air	10	26.1

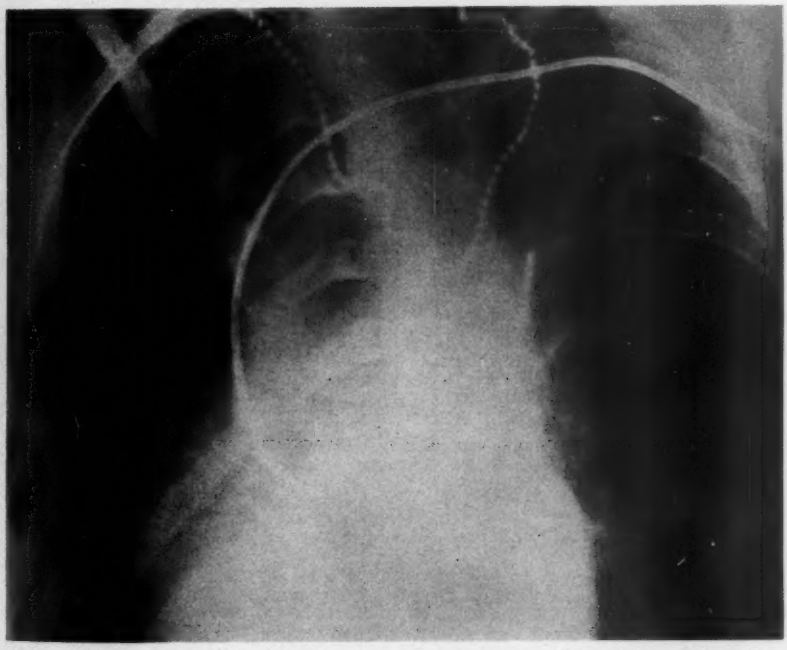

FIG. 11. Roentgenographic study showing catheter passing into the left auricle and into a pulmonary vein in a case of pulmonary valvular stenosis with intact interventricular septum.

for the fluoroscopic picture. Previous to that time the diagnosis had been tetralogy of Fallot.

Electrocardiogram: High and wide P-waves, right axis deviation, high RV_1, ventricular premature beats, $V_{2,3,4}$ inverted, and deep SV_4. This was interpreted as definite electrocardiographic evidence of myocardial disease compatible with right ventricular hypertrophy and auricular enlargement (fig. 14).

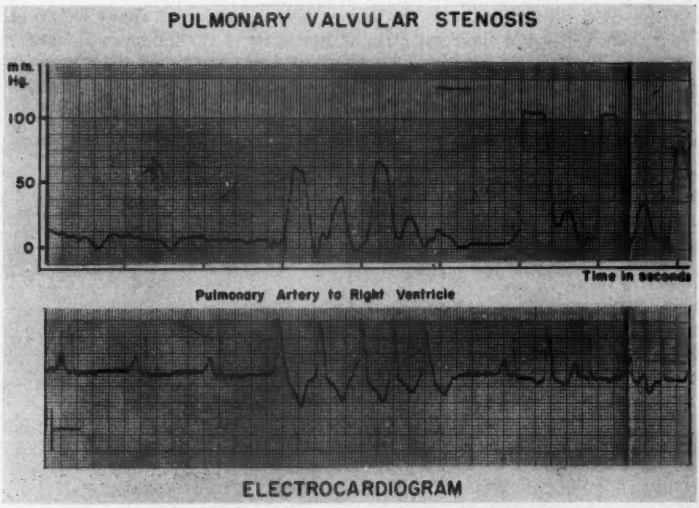

FIG. 12. Tracings of pressure as the catheter (fig. 11) is withdrawn from pulmonary artery into right ventricle.

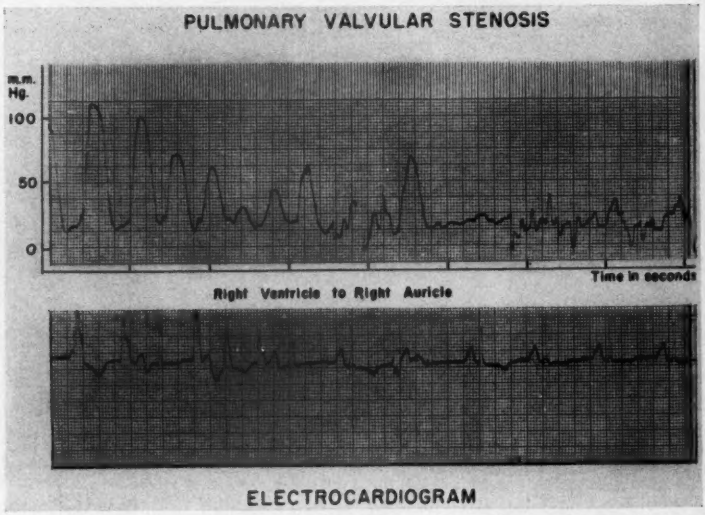

FIG. 13. Tracings of pressure as the catheter (fig. 11) is withdrawn from right ventricle into right auricle.

Cardiac catheterization was done on March 24, 1949 in the Heart Station at Charity Hospital, and the net right-to-left shunt was calculated[35,38] to be 46 per cent of the systemic venous return to the heart. No left-to-right shunt was demonstrated. The pressure tracings were not helpful because of a damping artifact in some of the tracings. A pulmonary artery pressure of 16.5/10 mm. Hg and a right ventricular pressure of 110/70 mm.

Hg were registered. In October 1951 arterial oxygen saturations at rest and during exercise with and without inhalation of oxygen were determined. The net right-to-left shunt was approximately 50 per cent of the venous return to the heart at that time (table 3).

Cardiac catheterization was repeated on Nov. 13, 1951 via the left antecubital vein (table 2 and figs. 12 and 13). The catheter passed from the right to the left atrium and on into the pulmonary vein where normally saturated blood was obtained (fig. 11). A pressure tracing taken with the catheter in the pulmonary vein showed a wave corresponding to atrial systole. After the catheter was passed into the pulmonary vein it was withdrawn and passed through the right ventricle into the pulmonary artery. The ventricular pressure was again found to be not only higher than the normal pressure but distinctly higher than the arterial systolic pressure taken simultaneously. The net right-to-left shunt was approximately 50 per cent of the systemic venous return to the heart at the level of the atria.

In view of these findings a diagnoses of pulmonic stenosis with interauricular shunt was made and the patient was advised to have an operation. She finally consented to operation which was done Nov. 21, 1951. In the waiting interval she began to have dependent

TABLE 3

Arterial oxygen saturation

Pulmonary valvular stenosis

	Percentage	
	Preoperative Oct. 25, 1951	Postoperative April 25, 1952
Air Rest	71	88
Air Exercise	61	86
O_2 Rest	87	99
O_2 Exercise	63	95
Net Shunt (Right to left)	50	15

edema, increasing exercise intolerance, dyspnea occasionally, pain in her right side after exertion, and her menstrual periods, which always had been irregular, ceased.

Operation: The operation was done as described above. Blood loss was negligible.

Postoperative Course: Prior to operation the hematocrit was 71, and by the eleventh postoperative day it had fallen to 43. Other than an infusion reaction with chills and fever, the patient's course postoperatively was not remarkable, and she was discharged on the thirteenth postoperative day.

Dependent edema following her discharge from the hospital necessitated her adherence to a salt-free diet after which the edema subsided. Aside from an occasional cold, her only other complaint was aching of the left side in the region of the scar.

After treatment by her family doctor with penicillin the patient was admitted to the hospital with fever two months after operation. Although tentative diagnoses of subacute bacterial endocarditis and pneumonitis were made, they were never proved. The patient was found to be sensitive to penicillin, and her fever subsided after the drug was discontimed. A rash, which appeared three days later, persisted several days. Reaction to the antibiotic could therefore account for the entire episode.

Since the last hospital admission the patient has had no major illnesses and few minor complaints. Pain in the region of the scar bothers her no longer, and her menstrual periods

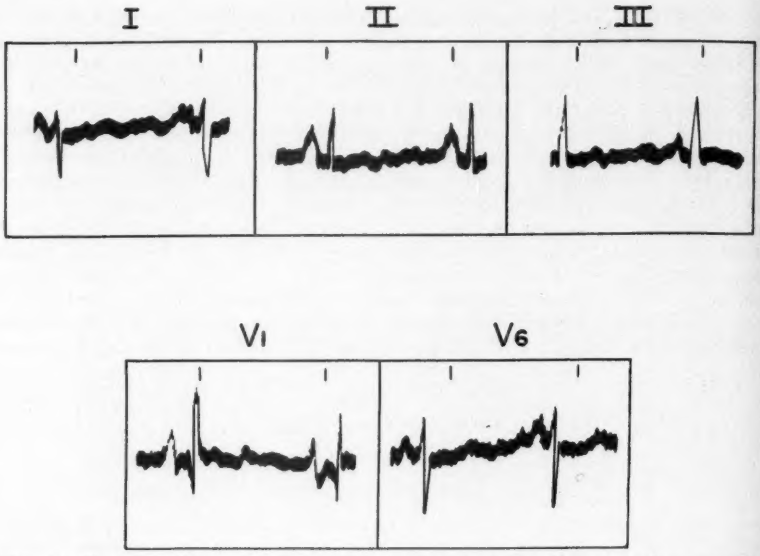

FIG. 14. Electrocardiogram in a case of pulmonary valvular stenosis before operation, showing large P waves and right axis deviation.

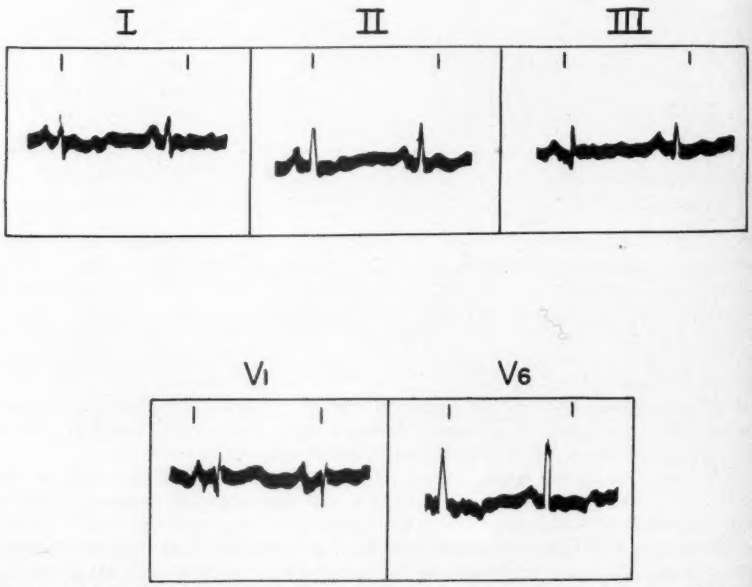

FIG. 15. Electrocardiogram taken after operation, illustrating regression of the abnormalities shown in figure 14.

have returned. Although cyanosis has not entirely disappeared, it is not noticeable, and the abnormal pigmentation of the skin and sclerae are no longer visible. Clubbing is not as pronounced as it was when she was first seen but it is still present to a degree. Arterial oxygen studies on April 25, 1952 give objective evidence that pronounced improvement has been achieved by the operation (table 3). The net right-to-left shunt at the atrial level at rest was between 10 and 15 per cent of the systemic venous return to the heart compared to 50 per cent preoperatively. The hemoglobin fell 6 Gm. and is now normal, 15.1 Gm., and the hematocrit has also remained within normal limits. Right axis deviation and P-wave changes noted in the electrocardiogram have also regressed (figs. 14 and 15) to a remarkable degree.

DISCUSSION

After any therapeutic procedure it is well to evaluate results as objectively as possible. Catheterization and arterial oxygen saturation studies offer an opportunity to do this, although their interpretation in relation to the clinical course is still in process of evaluation.[26, 33] In the case of mitral stenosis presented, pulmonary artery and pulmonary capillary pressures fell as did pulmonary arteriolar resistance. However, the extent of irreversible change in pulmonary vessels[39, 49] is a question which is difficult to answer on the basis of catheterization results, and clinical improvement may be greater than anticipated from pressure readings. Certainly in this case not only did the patient's clinical course and symptoms change for the better, but mitral commissurotomy resulted in objective evidence of decreased pressure in the pulmonary circulation.

The net shunt from right to left in the patient with pulmonary stenosis fell from 50 per cent to 15 per cent of the peripheral circulation as a result of operations. Other evidence of improvement was diminution in size of P waves and right axis deviation. Clinically, cyanosis was greatly alleviated. After the valve is open, improvement is apt to be very striking in cases of pulmonic stenosis with intact interventricular septum, and this case was no exception.

The occurrence of stenosis of the pulmonary valve in tetralogy is an additional reason to enter the thoracic cavity on the left side in these cases. A shunt is performed in cases with infundibular stenosis and valvulotomy is performed if valvular stenosis is encountered. We have adopted this approach in all routine cases of tetralogy of Fallot and inspect the pulmonary artery and infundibulum with the pericardium open more frequently than in the past. Further reports on results after direct relief of low infundibular stenosis are awaited with interest.

Valvulotomy for mitral or pulmonic stenosis carries with it the risk of a fatal outcome in any case, but the risk seems reasonable when weighed against the possible gain. Although results of catheterization are not always encouraging,[33] clinical and pathological studies,[12, 37] indicate that the leaflets remain open after these procedures and that the effect of operation is lasting.

SUMMARY

Although a bloodless field, adequate visualization, and ample time are desirable in intracardiac surgery, commissurotomy for mitral stenosis and valvulotomy for pulmonary valvular stenosis have emerged as acceptable closed procedures.

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ENDOMETRIOSIS

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The discovery of the transfer of fragments of endometrium from their normal location and of their implantation upon ovary, uterus and other structures is of much importance to the surgeon. It is an extremely fascinating condition, the only one in which benign tissue has the ability to invade other tissues. The disease occurs with such frequency that one marvels at the fact that it was unrecognized for so many years after pelvic surgery had become commonplace.

Cullen[3] gave the first definite clue as to the true significance of the origin of the islands of ectopic mucosa, when he proved that the glandular phases of adenomyomas were derived from the endometrium. The earlier authorities attributed them to adult remains of the wolffian system; inclusions of mullerian rests; the influence of inflammation; and metaplasia. Sampson's[8] implantation theory is now more widely accepted than any of the previous theories. Sampson evolved his theory after an exhaustive study of perforating hemorrhagic cysts of the ovary, and proved that misplaced endometrial tissue, wherever found, is a histologic structure identical with that of uterine mucosa, and that it is governed by the same physiologic factors. Uterine or tubal epithelium may escape into the pelvis during menstruation; lodge on the various pelvic structures (especially the ovaries); become implanted, and eventually develop into adenomatous growths that respond to the stimulus of the sex hormones, as does the normal endometrium. Sometimes the endometrium invades the myometrium and gives rise to endometriosis interna or adenomyosis.

Microscopically, the picture of endometriosis is one of well defined endometrial glands lined with columnar epithelium and surrounded by cellular stroma in variable amounts. The endometrial masses are called endometriomas and are filled with blood or pigment at menstrual periods. They pucker the tissues and form dense tough adhesions. These implants (fig. 1) are rarely found above the umbilicus but have been found primarily on the serosa of the pelvic viscera.

The distribution by organs in the private cases which we have had in the last five years agrees very closely with Table I. When adenomyosis is added to the external lesions of the uterus, however, then the ovarian involvement was surpassed in our series by that of the uterus. The implants appear as small cysts in the serosa, often bluish from contained blood. By rupture, secondary implants may result. On the ovary they tend to progressive growth and become the source of cystic ovarian tumors (fig. 2).

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The lesions of the ovary vary in size from small ones of pinhead size to those larger than a child's head.[10] The small superficial lesions are more common on the convex ovarian surface and vary in color from dusky red to brownish black, depending upon the condition of the contained blood. The larger cysts

FIG. 1. Endometrial implant on appendix. A rare lesion

TABLE I

Distribution of endometriosis by organs

Frequency of occurrence according to Haydon[4] in 569 cases

Location of Lesion	Frequency of Occurrence
Ovaries	343
Uterus	313
Cul-de-sac	191
Uterine ligaments	69
Tube	16
Rectum	14
Sigmoid	8
Abdomen	3
Bladder	3
Appendix	1

are often rather thick-walled and dull white in color, while in the thinner areas the dark chocolate-colored contents may darken the wall to brown or almost black. The cysts characteristically perforate when the pressure from intra-cystic hemorrhage becomes sufficiently great. Organization following this discharge of blood results in sealing the cyst either upon itself or to contiguous structures.

The peritoneum, irritated by this blood, becomes hyperemic and thickened. Upon opening the peritoneum the characteristic picture is a variable quantity of old blood which is free in the cavity. When the cyst is dissected free, the point or points of previous perforations are invariably broken into allowing the contents to escape. This usually consists of a thick brownish fluid from which the name *chocolate cyst* has been derived. The presumptive evidence is in favor of an endometrial cyst when, upon dissecting free an adherent ovary or ovarian cyst, chocolate or tarry material appears.

Other ovarian cysts, including corpora hemorrhagica, may give a similar gross picture. A distinguishing point is that the blood from the corpus hemorrhagicum is much more apt to be clotted. Also, hemorrhage into a serous cystadenoma may

FIG. 2. Endometrial cyst of ovary. A common lesion of endometriosis

give a chocolate color to the contents, but the blood, being mixed with serous fluid is not usually sufficient to form the characteristic thickness of the endometrial cyst contents.

Implants are common on the surface of the uterus, especially posteriorly and inferiorly. The cul-de-sac is a favorite place for endometrial implants; the posterior wall of the vagina often becomes densely adherent to the anterior rectal wall, thus completely obliterating the cul-de-sac. The uterosacral ligaments on either side of the cul-de-sac are commonly involved in the endometrial growth and form small, puckered shotty nodules along their course. An extension of the endometrial cells downward may result in invasion of the rectovaginal septum with involvement of the anterior rectal wall and at times penetration into the mucosa of the posterior vaginal vault. Rarely this growth may be visible through

a vaginal speculum as a dark nodular mass posterior to the cervix. Much more often, according to Stringer,[9] transillumination of the cul-de-sac and rectovaginal septum will disclose involvement.

Small endometriomas may occur on the serosa of the round ligaments. Cases have been reported of endometriosis of the umbilicus. Here the endometrial tissue infiltrates the surrounding tissues and by hemorrhage forms bluish nodules, sometimes visible through the skin. Laparotomy scars from operations upon the uterus and the tubes occasionally are the sites of endometrial growths.

CLINICAL ASPECTS

Endometriosis is characteristically a disease of reproductive life, occurring most frequently between 25 and 40 years of age. A fundamental point in the consideration and the management of endometriosis is that the aberrant endometrium, like that of the uterus itself, is under the physiologic and trophic control of the ovary. As long as functioning ovarian tissue is present, there is always the theoretic possibility of persistence and extension of the endometriosis. As a matter of fact, however, there are wide individual variations in the clinical significance of the condition.

At one extreme is a considerable group of cases in which the endometriosis is of greater scientific than clinical significance. Small areas of endometriosis may be accidentally found either at operation or upon routine laboratory examination of the uterus, ovaries or other pelvic organs, with no symptoms referrable to such lesions, and with little or no tendency to progress, as observed in the subsequent course of such patients. At the other extreme are cases in which the process extends rapidly with widespread involvement of the pelvic structures and with the production of severe symptoms. Even complete obstruction of the rectum or sigmoid may occur[5] as cited by us in reporting 2 such cases in 1944. Between these two extremes is the largest and clinically most important group in which, for example, one finds endometrial cysts of one or both ovaries, with or without moderate involvement of the uterosacral ligaments, rectum or other organs. These are the cases which often call for the exercise of careful judgment on the part of the surgeon.

According to Meigs and Sampson, 10 to 20 per cent of all gynecologic cases have endometriosis, as do 11 per cent of all women. The incidence of endometriosis in private gynecologic cases is 36 per cent, according to Meigs,[7] and only 8 per cent in charity patients. It is also true that the educated class has fewer babies and at a later age and more endometriosis because they practice better birth control.

The symptomatology (Table II) is not unlike that of chronic pelvic inflammatory disease, except that in many cases severe dysmenorrhea is present which is frequently referred to the rectum or sacro-coccygeal region. This is explained by the premenstrual and menstrual swelling of the uterosacral endometriomas often present.

No specific criteria for the diagnosis of endometriosis exist. The symptoms[1] produced vary so widely with the location, size, extent, and activity of the lesion

that identification depends upon eliciting and properly interpreting the presenting complaints in their relationship to the menstrual cycle and the pelvic findings. As ovarian stimulation produces proliferation and engorgement of the ectopic endometrium, the classic patient suffers from pelvic pain during the days preceding menstruation. This pain, fulminating with the flow and continuing throughout its duration is usually interpreted as dysmenorrhea. Pelvic comfort does not return for several days. This dysmenorrhea, once established, becomes more severe with each succeeding period. It is characteristic of this pelvic pain that heat, so soothing to most pelvic discomforts, by increasing engorgement aggravates its severity; and that antispasmodics are of but little benefit.

To this general picture may be added both menorrhagia and metrorrhagia from uterine adenomyosis, tenderness in the posterior fornix and dyspareunia produced by active lesions in the cul-de-sac, and both dyspareunia and dyschesia by involvement of the rectovaginal septum. If the bladder wall is invaded, frequency of urination and dysuria result; if the intestinal wall is invaded, symptoms vary from slight digestive disturbances to those due to complete obstruction. When extensive areas of the pelvis are involved, the massive firm adhesions may produce constant discomfort, there being a periodicity to the pain only if the lesions remain active. When pressure within a cyst becomes sufficient to suppress or destroy the endometrium, activity ceases; and lesions, of such size as to produce a bulging of the abdominal wall, become asymptomatic. Endometrial ovarian cysts which have been previously silent may rupture, producing the symptoms and signs of acute peritonitis. Since the symptoms of endometriosis vary widely in their scope and are produced individually by many pelvic conditions, the classic case is rare. Therefore, it is only by the proper correlation of the symptoms with the menses and accurate interpretation in terms of the pelvic findings that an accurate preoperative diagnosis can be made. To make the diagnosis of endometriosis even more difficult is the fact that it may be found associated with every other lesion of the pelvis including carcinoma.

TABLE II

Symptom complex of endometriosis

1. Age between 25 years and the menopause.
2. Sterility, absolute or relative.
3. Abnormal menstruation, usually menorrhagia.
4. Acquired dysmenorrhea.
5. Dyspareunia.
6. Sacral backache.
7. Intermenstrual lower abdominal pain with increased discomfort at the time of menstruation.
8. Pain in the rectum or bladder which bears a distinct relationship to menstruation.
9. Constipation may at times mean invasion of the bowel with partial obstruction.

The findings on examination vary considerably. Endometrial ovarian cysts are often present. They may be bilateral and are usually fixed. Not infrequently the

fundus is retroposed and adherent. Fibroids may be present. In the posterior cul-de-sac often there is a nodular area of induration. This may extend laterally into the region of the utero-sacral ligaments and may cause fixation of the rectum. Rectal, and vagino-rectal examinations are very helpful in these cases.

In the absence of evidence suggestive of pelvic infection, an ovarian cyst with either an associated fixed retrodisplacement of the uterus or/and with one or more nodules in the uterosacral region, speaks strongly for the presence of endometriosis.

TREATMENT

The prophylactic treatment of endometriosis would seem to be early marriage and early childbearing. In addition, it seems advisable not to do pelvic examinations, tubal inflation tests, curettages, or other intrauterine manipulations during or near menstruation. We have three major weapons to use in the active treatment of endometriosis: surgery, irradiation, and hormones; certainly testosterone and possibly stilbestrol.

Since a positive diagnosis of endometriosis may be difficult,[10] surgical exploration is generally preferable to treatment with irradiation done blindly. This is particularly important when ovarian enlargement and/or nodular induration make differentiation from malignancy difficult. Surgery is also to be preferred to irradiation because many of the symptoms are frequently due to mechanical factors such as adherent retrodisplacement of the uterus, pressure from large chocolate cysts and intestinal obstruction. Also, other pelvic lesions, particularly myomas, frequently accompany endometriosis, and their surgical removal is desirable.

The treatment of ovarian endometriosis in the early stages of the condition should be as conservative as possible; due regard being paid to the future fertility of the patient and also to her need of ovarian hormone. However, when both ovaries are reduced to the state of thin walled adherent hematomas they must be removed.

Because the disease so frequently manifests itself in relatively young women, the choice of proper treatment may be extremely difficult. Whenever operation is contemplated for endometriosis, the patient should be advised of the possibility of radical surgery. For the final decision must be made at the operating table where the extent of the disease can be carefully determined. Because of the possibility of conserving the menstrual and childbearing functions in young women, surgical treatment is definitely preferred to castration by irradiation in this group.

In debating conservative versus radical surgery at the operating table, each case should be judged individually, taking into consideration the extent and the location of the lesions, the patient's age, her nervous constitution and her feeling concerning pregnancy. In women over 40 years of age, a large percentage should be treated radically, and, when in doubt, it is probably better to err on the radical side. But even in this age group a perfectly normal ovary may be saved at times.

Another point to be considered in cases of endometriosis is whether or not the

uterus should be removed at operation. After 40 years of age, hysterectomy should be practically an invariable rule when laparotomy is done for endometriosis. In young women in whom childbearing is extremely vital, we have gone to extremes to retain sufficient remains of the pelvic organs to preserve this function. And in some instances we have been rewarded by future pregnancy when as little as one-half of one ovary was saved. Occasionally endometriosis is present on the posterior surface of a retroverted uterus in a young woman with perfectly normal ovaries. Such a uterus should be freed, the implants fulgurated and a uterine suspension done. Counsellor[2] recommended pre-sacral neurectomy in such cases so that, in the event of recurrence, no dysmenorrhea or dyspareunia would result.

Usually endometrial lesions involving nonadnexal pelvic structures may be easily and safely excised and ovarian conservation practiced. If radical ovarian surgery is done, local excision may not be necessary. In the patient near the menopause, an extensive lesion of the sigmoid causing obstruction can be treated satisfactorily by bilateral oophorectomy with colostomy if necessary. In younger women, however, a segmental resection of the involved portion of colon without oophorectomy is the less radical procedure.[6]

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CONGENITAL ESOPHAGEAL ATRESIA WITH TRACHEO-ESOPHAGEAL FISTULA

REPORT OF FIVE CASES

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Congenital esophageal atresia with tracheo-esophageal fistula is an uncommon congenital abnormality which is incompatible with life. This lesion is frequently associated with other congenital abnormalities.

It is reported that congenital atresia of the esophagus occurs once in about 2,500 births. To date, over 450 cases of congenital esophageal atresia with tracheo-esophageal fistula have been reported.

Three types of atresia of the esophagus with tracheo-esophageal fistula occur. Vogt[5] has classified congenital atresia of the esophagus into three main divisions: 1. Agenesis of the esophagus. 2. Simple atresia of the esophagus. 3. Atresia of the esophagus with tracheo-esophageal fistula. The latter is divided into three types: Type A—Fistula between the proximal esophagus and the trachea. Type B—The proximal esophagus ending in a blind pouch with the distal segment communicating with the trachea or main bronchus. Type C—Both proximal and distal segments of the esophagus communicate with the trachea (fig. 1).

The first successful direct operative procedure to correct this anomaly was performed by Cameron Haight[4] of the University of Michigan in 1941.

It is thus obvious that previous to 1941 all of these infants died. Indirect operative procedures have been far from ideal, and in most instances a direct operative plan can be carried out.

DIAGNOSIS

A diagnosis of congenital esophageal atresia is not difficult. It should be suspected in any newborn with excessive mucous in the throat who is having attacks of choking, coughing, and cyanosis. Regurgitation following nursing is characteristic and may be associated with considerable respiratory distress.

After a clinical diagnosis is made, there are several confirmatory procedures that aid in determining the type of lesion present. A roentgenogram of the chest and abdomen will reveal air in the stomach if there is a fistula between the distal segment of the esophagus and trachea. In the absence of air in the stomach two possibilities exist. The patient may have a fistulous communication between the proximal segment of the esophagus and trachea, or there may be a complete atresia of the esophagus. The passage of a small catheter into the esophagus will readily reveal an obstruction. The instillation of 2 cc. of lipidol will demonstrate a blind pouch or esophago-tracheal communication between the proximal

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segment of the esophagus and the tracheo-bronchial tree. Barium should not be used since aspiration of this material will result in severe pneumonitis. Endoscopy is not necessary and may be dangerous from the standpoint of perforation or resulting laryngeal edema due to instrumentation.

When the diagnosis has been established, definitive surgical intervention is imperative. This should be done as soon as the patient's metabolic condition will permit. The presence of atelectasis can usually be corrected by aspiration of the tracheo-bronchial tree. The fact that the patient has a pneumonitis is no contraindication to surgical treatment.

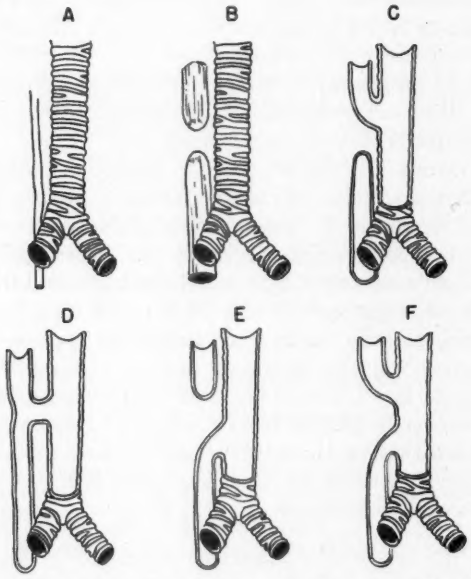

FIG. 1. Illustration revealing the various types of esophageal atresia and esophageal atresia with tracheo-esophageal fistula. The types vary from a complete atresia of the esophagus without fistula to tracheo-esophageal fistula without esophageal atresia.

TREATMENT

All patients having esophageal atresia with tracheo-esophageal fistula are hopelessly doomed to die without some type of surgical correction. Indirect operative procedures to circumvent this anomaly, employing staged operations, are indicated only in rare instances when it is impossible to do end to end esophageal anastomosis or gastro-esophagostomy.

The direct plan of approach is one in which the fistula is ligated and the continuity of the esophagus re-established by end to end anastomosis. Several variations of end to end anastomoses have been described. The side of approach is variable. Haight employed the left extra-pleural approach. We prefer a right posterior extra-pleural approach since better exposure is obtained. These in-

fants will tolerate an extra-pleural operation far better than a transthoracic procedure.

The operation is begun under local anesthesia and after the esophagus and tracheo-esophageal fistula have been exposed it is well to supplement the local anesthetic with a general anesthetic, such as cyclopropane, in an effect to avoid excessive mediastinal motion and retraction of the esophagus when the infant cries or swallows.

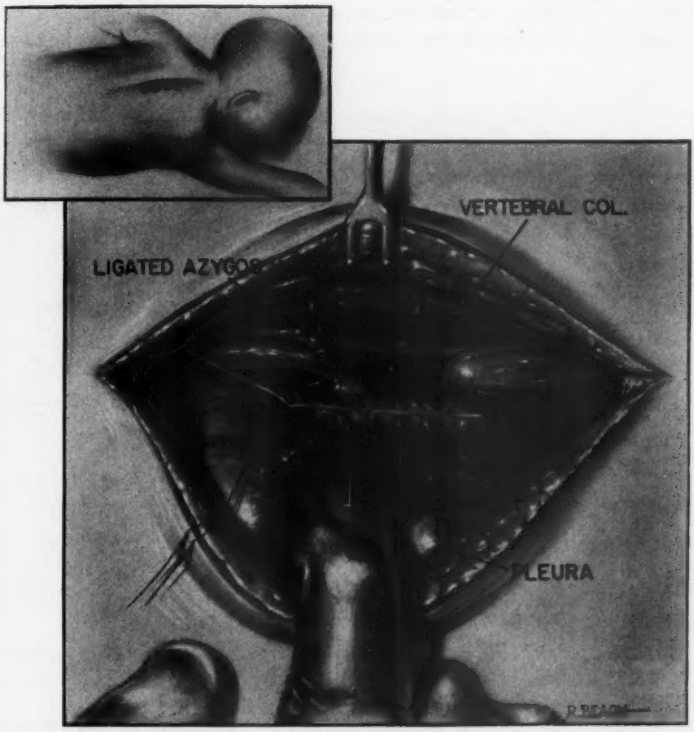

FIG. 2. Illustrates position of patient and the operative approach. This type of lesion is the anomaly most frequently encountered.

With the patient lying on the left side, a right paravertebral incision is made between the scapula and the spine for a distance of 6 cm. The rhomboid and trapezius muscles are clamped, divided, and ligated. A subperiosteal resection of the posterior portions of the third, fourth, fifth, and sixth ribs is done for a distance of 3 to 4 cm. Intercostal vessels, nerves, and muscle bundles are clamped, divided, and ligated. Blunt dissection of the parietal and mediastinal pleura is done with care to avoid perforation of the pleura causing a pneumothorax. Should the pleural cavity be opened inadvertently it should be closed

at once and the air aspirated. The pleura should be protected with a moist gauze sponge which is held in place with a delicate retractor. In most cases it is necessary to divide the azygos vein to further increase the exposure. The proximal and distal esophagus are then visualized and the tracheo-esophageal fistula identified (fig. 2).

The vagus nerve is injected with novocaine to avoid vagal stimulation which may result in cardiac irregularities or arrest. Traction sutures are placed in the end of the proximal esophagus. The tracheo-esophageal fistula is ligated close to the trachea with two black silk ligatures and the atretic portion of the esophagus is excised and traction sutures are then placed in the distal esophagus.

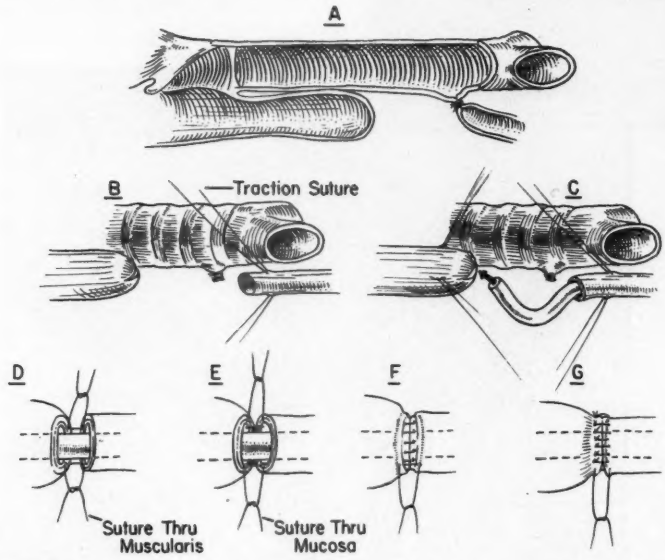

FIG. 3. Illustrative of operative technic with end to end anastomosis of esophagus over a stomach tube.

A no. 8 French catheter is passed into the distal end of the esophagus and allowed to enter the stomach. The catheter is then passed through an opening made in the proximal segment of the esophagus and the catheter is brought out through the nostril. The catheter is anchored to the mucosa of the distal segment of the esophagus with a fine black silk suture and an end to end two-layer anastomosis of the esophagus is made employing fine black silk sutures. Slight traction may be applied to the catheter in an effort to relieve any tension present on the anastomosis site (fig. 3).

A small Penrose drain is placed in the mediastinal space in proximity to the anastomosis. The wound is closed in layers using no. 000 chromic catgut. Interrupted black silk sutures are used for the skin. The retro-pleural space is allowed to completely re-expand before the wound is closed.

Antibiotics and oxygen are given and after 24 hours the child is fed through the inlying gastric tube. Frequent cleansings of the throat by suction is necessary in order to avoid aspiration of mucous (fig. 4).

The drain from the posterior mediastinal space is usually removed in four to to five days. At the end of 10 days a recheck on the anastomosis site is made by allowing the infant to swallow lipiodol. If there be no fistulous communication or stricture the tube is removed and the patient is started on a nursing schedule.

Between the years 1948 and 1951 5 proved cases of congenital esophageal atresia with tracheo-esophageal fistula have been observed in the Spartanburg General Hospital.

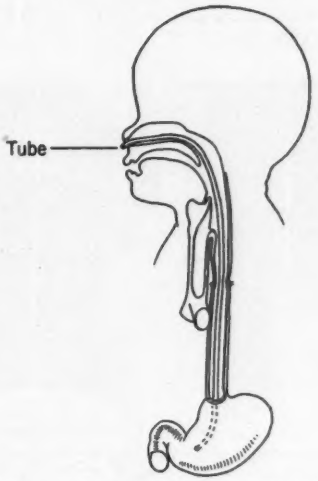

FIG. 4. Illustrative of completed anastomosis with inlying gastric tube

CASE REPORTS

Case 1. B. G. P. White female born on June 1, 1948. This patient had a classical history and clinical findings of esophageal atresia with tracheo-esophageal fistula. The diagnosis was made on June 10 by employing roentgenographic studies. The right extra-pleural posterior operative approach was made with closure of the fistula and end to end anastomosis of the esophagus over a tube. The patient died the following day as a result of pneumonia and pulmonary edema. No autopsy was obtained.

Case 2. B. B. C. Colored male born on May 9, 1948 at home. He was admitted to the hospital on May 11. A clinical diagnosis was made at that time on the basis of classical history and findings. The recommended roentgenographic studies and treatment were accepted on May 13 and the patient was operated upon the following morning. A right extra-pleural posterior approach was made. The patient developed cardiac arrest while the anastomosis of the esophagus was being performed. It was believed that this was probably due to vagal stimulation. No autopsy was obtained.

Case 3. B. B. L. White male born on March 4, 1949. There was classical history and findings of congenital esophageal atresia with tracheo-esophageal fistula. Clinical diagnosis was made on March 19 and confirmed by roentgenographic studies. Operation was done

on March 20 through a left transthoracic approach. The patient died on the operating table following ligation of the fistula. An autopsy was obtained. The cause of death was attributed to inanition and pneumonia.

Case 4. B. G. P. White female born on Dec. 3, 1951. There was a classical history and findings of esophageal atresia with tracheo-esophageal fistula. A clinical diagnosis was made on December 9 and confirmed by roentgenogram on the following day. The patient was not operated upon and died on December 11, apparently as a result of tracheo-bronchial aspiration. The patient was a premature baby.

Case 5. B. G. H. White female born on Dec. 25, 1951. A clinical diagnosis was made on December 27 and confirmed by roentgenographic studies the following day. The patient was operated upon on December 28. A right extra-pleural posterior approach was used. The fistula was ligated and an end to end esophageal anastomosis was made over a catheter. The vagus nerve was injected with novocaine preliminary to manipulation about the esophagus in an effort to avoid cardiac irregularities or cardiac arrest.

The patient made a relatively uneventful recovery (fig. 5).

The author operated on Cases 2 and 5.

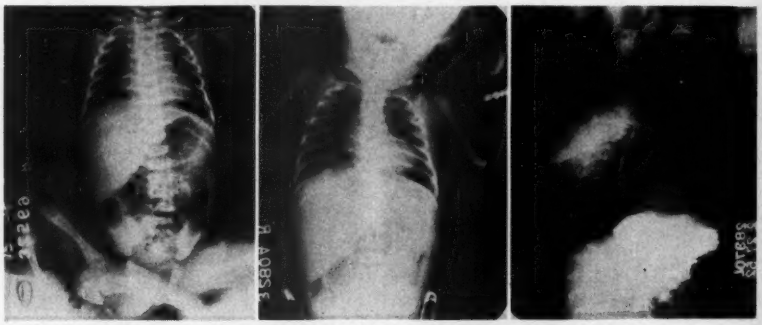

FIG. 5. Roentgenographs are illustrative of case 5. (A) A routine roentgenograph of the abdomen and chest. Note gas in stomach and also the congenital vertebral body defects. (B) A postoperative roentgenograph showing the rib defects and the inlying gastric tube. (C) A barium roentgenograph study taken eight weeks following surgery and reveals a relatively normal esophagram.

DISCUSSION

Since 1948 5 proved cases of congenital esophageal atresia with tracheo-esophageal fistula have been diagnosed in the Spartanburg General Hospital. Three cases occurred in females and 2 in males. All were white except one—a Negro boy.

Four cases were operated upon with only 1 patient surviving. The 1 surviving was diagnosed early before starvation and pneumonia had developed. One case was not given the benefit of surgery.

All patients presented a classical history and clinical findings of congenital esophageal atresia with tracheo-esophageal fistula. There was considerable delay in making a positive diagnosis or in initiating treatment. In the 5 cases an average of 7.4 days elapsed before a correct diagnosis was made, the longest being 15 days and the shortest 2 days. In all cases an accurate diagnosis was established by roentgenographic study.

Two cases occurred in 1948, 1 in 1949, and 2 in 1951. In 1951 there were a total of 3,006 births in the Spartanburg General Hospital and 66 stillbirths.

There were 14,143 admissions. Congenital esophageal atresia with tracheo-esophageal fistula comprised .014 per cent of all hospital admissions in 1951 or an incidence of 1 case in about 1,500 births. All cases were of the type 3-B in which the proximal segment of the esophagus ended in a blind pouch and the distal segment communicated with the trachea.

Previous to the past decade no known satisfactory operative procedure had been devised to correct this anomalous condition. All infants born with this condition died. It is imperative that the diagnosis be made early before the patient develops pneumonia and atelectasis or before marked metabolic derangement has occurred.

The cause of death in most of these infants is from pneumonia due to aspiration of saliva and milk from the blind end of the esophagus or through the fistulous communication between the proximal end of the esophagus and trachea.

Aspiration of gastric juice through a fistula between the lower segment of the esophagus and tracheo-bronchial tree also results in severe pneumonitis. Starvation with fluid and electrolyte imbalance is still another cause of death in these infants.

In all infants presenting this anomaly a careful search for other anomalies should be made since it has been shown that imperforate anus, club feet, absence of the kidneys, hydronephrosis, spina bifida occulta, rectovaginal fistula, hypospadias, hydrocephalus, and other anomalies occur. Case 5 had anomalies of the cervical and upper dorsal vertebrae. Brown and Brown[1] have found associated congenital anomalies in 8 out of 21 cases of esophageal atresia.

Gross and Scott[3] prefer to do an oblique to oblique anastomosis of the esophagus. It appears that this type of anastomosis may add tension to the anastomosis site. Daniel[2] invaginates the distal esophagus into the proximal esophagus with a catheter anchored to the distal segment for traction. This type of anastomosis is prone to cicatrization and esophageal stenosis.

In those cases where there is a complete atresia of the distal esophagus an esophagogastrostomy may be made or one of the circumventing stages operations done with construction of an external esophagus. Should one use a right posterior extra-pleural approach and find that the distal esophagus is atretic the wound should be closed after the fistula had been ligated and cervical esophagostomy should be made. In such cases a gastrostomy should be made for feeding. At a later date an external esophagus may be constructed.

SUMMARY AND CONCLUSIONS

Congenital esophageal atresia with tracheo-esophageal fistula is an uncommon condition. It is reported to occur once in 2,500 births, giving it an incidence similar to that of harelip. All infants with esophageal atresia are doomed to die unless early diagnosis is made and prompt definitive treatment is instituted. There is no need today to take a defeatist attitude toward this lesion. More and more satisfactory results are being reported and the operative procedures are well standardized.

Five cases of tracheo-esophageal fistula are reported. Four patients were operated upon with only 1 infant surviving. In this case the diagnosis was made early and surgical treatment was instituted without delay.

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HERNIATION OF LUMBAR INTERVERTEBRAL DISKS IN COAL MINERS*

A PRELIMINARY REPORT

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Since the classic description of the rupture of intervertebral disks by Mixter and Barr in 1934,[9] progress in the treatment of these lesions has been remarkable. In the last decade a voluminous literature has developed dealing with nearly all aspects of the intervertebral disk problem. So far as we have been able to determine, however, there has been no report of a large group of patients with lumbar disk lesions, all of whom were bituminous coal miners. We therefore present a preliminary report of such a group.

Our study is based upon an experience with 564 patients upon whom we have operated since 1946 for herniation of one or more lumbar intervertebral disks. It deals specifically with 303 of these patients who were coal miners. Their average age was 36 years, the youngest being 19 and the oldest a retired miner of 79 years. Most of these miners had been doing one of the many jobs in the mine or around the tipple or drift mouth on the outside before their first symptoms occurred. Miners of bituminous coal generally have the traditional background, outlook and physical stamina which make them suitable for hard labor, often under ground in cramped quarters. Some of these men worked at loading coal while on their knees or in stooped positions in coal tunnels no higher than 26 to 40 inches. Others laid ties and rails and prepared track for the coal cars or placed heavy timbers on top of upright pilings to support the tunnel roof. Still others handled heavy equipment, ran motors and coal cutting machines, handled *duck bills* or used explosives for shooting coal. These are laborious tasks, permitting repeated trauma to the low back, which has been considered productive of the disk cracking and degeneration preceding herniations.[3, 4, 5]

Schmorl, who based his opinion on the investigation of 8,000 spines, is quoted by Lewey[5] as stating, "As I become more familiar with disks and their pathologic changes, I am more and more convinced that their tissue is most delicate and vulnerable. It is frequently damaged by strain, almost within the range of normal movement or by minor external force." Lewey[5] stated further, "Anatomic studies indicate furthermore that the annulus fibrosus is less dense posteriorly than anteriorly and laterally, and finally that the disk itself is less firmly fixed in the bone at the posterior circumference of the vertebra. If we combine these considerations with the normal architecture of the disk lamellae it is easily understood that mild torque of the spine may lead to rupture of lamellae of the disk with subsequent necrosis and possible liquefaction."

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The coal miner subjects his back to that torque and strain repeatedly while at work, yet only 69 of our patients had their onset of symptoms so definitely associated with work injuries as to be accepted as compensation cases. We would stress, therefore, that the coal mining industry has by no means a monopoly on the production of herniated disks. The other 261 of our lumbar disk patients were in other walks of life, many with sedentary occupations, and one man sustained a complete and sudden extrusion of a lumbar disk sequestration at lumbar 4 when he quickly jumped up from sitting in a chair.

Lest we be criticized for excessive surgery[1, 2] we emphasize that all of these patients upon whom we operated for ruptured disks had first received at our hands, or under the care of the referring physician, one or more courses of conservative treatment without relief of their symptoms. Our method of conservatism consisted of complete rest on a hard bed, daily physiotherapy with infra-red ray, diathermy or hydrogalvanism to the back and legs, salicylates or Tolserol in full doses by mouth and occasionally leg traction, followed by a plaster of Paris jacket or high lumbar belt for ambulation. Steindlers test injection, (as described by Hyndman[4]) with novocaine or nupercaine in oil was done to rule out a myofascial origin for the pain when indicated. We learned early in the series not to urge operation upon these patients and now seldom do so. It was the patient who usually tired of conservatism and requested operation. Economic pressure due to loss of work and pay caused some patients to request operation early in their hospitalization.

A history of trauma was elicitated in 119 cases and in 184 no direct history of trauma was obtained. The type of injury was recorded as follows:

Fall	9
Twist	5
Struck by object	8
Lifting injury	17
Strain or sprain	27
Injury to back at work	32
Miscellaneous	19
Jackknifed	2

The onset of pain was sudden in 140 patients and gradual in 163. The site of onset was in the back in 170, in the involved leg in 128 and unrecorded in 5 patients. Rarely the first pain was said to have occurred on the side opposite that which later became constantly painful and on which the leg was involved. Usually when the pain began in the back it was confined to the lumbosacral or gluteal region for a time and then spread down one of the legs to the peripheral distribution of the involved nerve root below the knee.

All of the patients had a preliminary general physical examination with a blood count, urine examination and serology tests made. No correlation of significance in any of these findings with the disk pathology was possible. Of the neurologic findings, the site of hypoesthesia and associated muscle weakness or atrophy, reflex changes when present, and the location of percussion tenderness at the flexed spine or its point of radiation down the leg were of greatest value in

determining the site of the lesion. List of the spine, limitation of straight leg raising, pain on the side of the lesion from straight leg raising on the opposite side, limitation of forward and backward bending, and inability to walk on heels or toes were found to be significant helps in diagnosis. Lumbar punctures were done on 253 patients and early in the series we believed that an increase in spinal fluid protein above our normal 15 to 25 mg. per 100 cc. was significant, particularly if these were 5 or 10 mg. more in the first than the second tube. Later we came to use the spinal puncture primarily to test for manometric abnormality and to help rule out nondisk lesions. Our highest protein reading was 100 mg. per 100 cc. and the lowest 5 mg. per 100 cc. in true disk herniation. Roentgenograms were made of the lumbosacral spine in all cases. Findings which we considered significant, if they coincided with other signs, were irregularities in the articular facets, narrowing of the intervertebral space, marked flattening or reduction of the lumbar lordosis and lipping or hyperostosis about the suspected intervertebral space. However, definite correlation of the roentgenologic findings with the disk lesion in our patients was so unsatisfactory that we have come to look to the roentgenogram as a means of helping to rule out lesions of the bone *per se* and only as an adjunct in disk diagnosis. In 5 selected patients myelography was done with pantopaque and in 39 with air; 26 were positive, showing clear cut defects and 15 were negative. Of the patients who had pantopaque myelograms, one of the two negatives was later found to have a disk herniation at lumbar 5, and all of the positive findings were verified (fig. 1). Of the air myelograms two of those reported positive were inaccurate as to the level of the lesions

FIG. 1. Herniated disk, central pantopaque defect, lumbar 4 in patient with bilatera nerve root signs.

and in 1 reported negative the patient was later proved to have a disk lesion. Only 2 patients had disk lesions definitely associated with a fracture of a lumbar vertebra (fig. 2). Discography and injection of a suspected intervertebral disk with roentgenologic opaque substance has been reported and in some instances recommended as a diagnostic procedure.[6, 7, 11, 12] No discography or injection of the disk was done preoperatively in any of our patients. This decision was made because of our belief that until proved otherwise, any injection into the disk substance must be considered a procedure potentially capable of initiating rupture and fragmentation of a normal disk.

Before operation the procedure was explained to each patient and an attempt was made to reassure him. His hospitalization status was established; his period

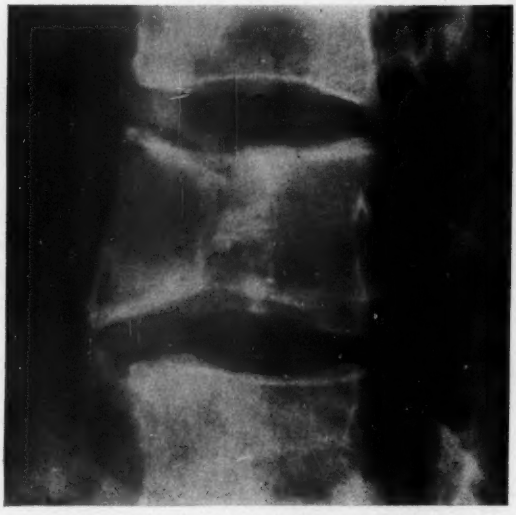

FIG. 2. Fracture lumbar 3, lipping lumbar 3–4. Aerogram with bulge of disk, Lumbar 3

of convalescence was discussed; and the dangers, possible complications, and expected benefits evaluated as accurately as possible. Mild sedation and an enema, if needed, were prescribed the night before operation. Three grains of nembutal were given one hour before operation. Scopolamine, atropine, and pantopon in the usual dose were given when the patient was brought to the operating room. Three patients were operated upon at their request under general anesthesia. The remainder were given spinal anesthesics with procaine. There were no deaths and no significant problems in anesthesia. Any fall in systolic blood pressure below 100 mm. of mercury was treated by the use of intravenous 5 per cent glucose in water, occasionally supplemented by small quantities of levophed.

In all cases, when feasible, a unilateral paravertebral incision through the

erector spinae fascia was used and usually a partial hemilaminectomy[8] was done to expose the disk herniation. This required only minimal enlargement of the intervertebral space without disturbance of the articular facets. We did not hesitate, however, to make a larger opening if this enabled us to remove the disk herniation with less traction or displacement of nerve structures. The ligamentum flavum was always removed from the nerve foramen as well as from the operative site, and a search was made for wandering disk material in each case with a nerve hook. Disks were considered pathologic when there was extrusion of disk substance, marked bulging against the root, or the annulus was so thin that movement of the vertebra caused bulging or the Cushing forcep met only slight resistance before penetrating into the softened disk. These pathologic disks were removed as completely as possible with the pituitary punch and curette. One patient had the removal of the disk followed by primary fusion by the orthopedist for marked spondylolisthesis. One patient had a secondary fusion because of instability. All closures were made without drainage.

Findings at operations were as follows:

	L 5	L 4	L 4 & 5	L 3, 4 & 5	Others
Disk Herniations					
Left	51	2	83	9	
Right	32	11	87	8	
Bilateral	2	2			
Cyst on Nerve Root					
Right	1				
Hypertrophy Ligamentum Flavum					
Right					1
Left					1
No Pathology Found					11

Postoperative care consisted of diet as tolerated, sedation with pantopon for 24 hours, enema on the third day if necessary and catheterization only if voiding could not be accomplished by the patient standing at the side of the bed or with the use of pressor drugs. The average stay in bed was 6.1 days and during the last half of the series, our routine was 5 days in bed and then up, except, of course, when there was a contraindication to ambulation. One patient was in bed 20 days and one man refused to stay in bed longer than the first day after operation. Postoperative instructions stated that there was to be no lifting, straining or driving an automobile for one month. Calisthenic exercises were then begun. Physiotherapy was used if it helped in rehabilitation. Occasionally plaster of Paris jackets or high lumbar belts were prescribed for patients with multiple disk lesions. A hard bed was used for 3 months.

Return to lighter occupations such as watchman, checker, weigher and lamphouse tender was allowed at the end of two months. Return to heavy manual

labor was permitted at the end of three months or as soon thereafter as the patient was capable. Complications were as follows:

Deaths	0
Footdrop	2
Infection, mild	3
Subarachnoid hemorrhage	1
Sacral neuritis and bladder symptoms	1
Pain requiring reoperation and finally prefrontal lobotomy	1

This last patient who required the prefrontal lobotomy before returning to work had been operated upon twice elsewhere and, upon re-exploration, we found still further disk herniation and extensive adhesions. Because of the obvious residual pain as well as some mental aberrations, the lobotomy was done and the man was rehabilitated, and returned to work at the mine. One patient upon whom we had done a full left fifth lumbar hemilaminectomy with removal of a herniated disk developed signs of cauda equina pressure after returning to work. At reoperation by Dr. Tice in Roanoke[10] the remaining right half of the lamina was found to have fractured and compressed the cauda equina. Removal of the fractured bone relieved the symptoms. Concerning the other complications, the infections in all 3 patients healed within one month; the patient with the subachranoid hemorrhage continued to have some symptoms and was not well rehabilitated although he returned to work; of the 2 patients with postoperative footdrop, 1 still has 50 per cent weakness which we attribute to the use of the coagulating current too near the root; and the other patient's condition became normal before three months. The patient with the sacral neuritis has a normal spinal fluid and little or no pain, but still has perineal hypoesthesia, loss of libido and incomplete bladder emptying. After 10 months his symptoms are clearing slowly.

According to the records, 277 of these patients had returned to work at the mine or were convalescing well at the last follow-up visit. Twenty-six patients had not resumed mining or other work due to causes such as low back weakness and discomfort, inability to stoop and bend comfortably, fear or dislike of mining, or eligibility for a pension. A few still had compensation ratings pending. Eleven miners have returned for reoperation due to recurrence at the same or different levels.

SUMMARY AND CONCLUSIONS

We have made a preliminary report of 303 coal miners who were operated upon for herniated lumbar intervertebral disks after conservative treatment had failed to relieve their symptoms or reveal other causes for their complaints.

Thirty-five and nine-tenths per cent claimed direct injury as a cause of their complaints but only 22.7 per cent were considered to be work injuries. One hundred and eighty-four patients, or 60 per cent, gave no specific history of injury.

Thirty-three per cent of patients had single lesions, 56 per cent had two disks involved and 5.6 per cent had three pathologic disks. This is a rather high percentage of multiple disks and it suggests that many of the patients with single

disk lesions may have become symptom free with conservative treatment. Three and six-tenths per cent revealed no disk pathology at operation to explain both backache and sciatica, notwithstanding repeated preoperative attempts to rule out other etiology.

Plain roentgenograms and laboratory tests were not diagnostic and we found that the diagnosis of lumbar intervertebral disk herniation had to be made primarily by the neurologic examination supplemented in 14 per cent of the cases by myelography.

Two hundred and seventy-seven of our patients were recorded as having returned to work or convalescing satisfactorily at last follow-up. A report on their work records and ability to remain at work, together with a later complaint and morbidity report must await the analysis of a five year follow-up which is intended.

It is our belief at this time that miners with true disk herniations, who have not been relieved by conservative treatment, should be operated upon. It is further our opinion that the present type of operative procedure is medically and economically sound. We believe that the majority of the lumbar disk patients can then return to work at the mines, but that most miners who have had multiple disk lesions should not work in extremely low coal seams.

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